

PUBLIC REVIEW DRAFT

**BASELINE HUMAN HEALTH RISK ASSESSMENT
VASQUEZ BOULEVARD AND I-70 SUPERFUND SITE
DENVER, CO**

July, 2000



Prepared for
US Environmental Protection Agency, Region VIII
999 18th Street, Suite 500
Denver CO 80202



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ISSI Consulting Group, Inc.
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EXECUTIVE SUMMARY

1.0 BACKGROUND

1.1 Site Description

The Vasquez Boulevard and I-70 (VBI70) Superfund Site is an area of approximately four square miles located in the north-central section of Denver, Colorado. The site is composed of a number of neighborhoods that are largely residential, including Swansea/Elyria, Clayton, Cole, and portions of Globeville. Most residences at the site are single family dwellings, but there are also some multi-family homes and apartment buildings. The site also contains a number of schools, parks, and playgrounds, as well as a number of commercial and industrial properties. Figure ES-1 is a map which displays the site.

1.2 Basis For Potential Concern

The site came to the attention of the U.S. Environmental Protection Agency (USEPA) because studies directed by the Colorado Department of Public Health and Environment (CDPHE) at a nearby site (Globe Smelter) indicated that elevated concentrations of arsenic and/or lead occurred in the soil of some residential properties in the Swansea/Elyria area. The source of these elevated levels is not known, but *a priori*, it is considered plausible that the contamination is associated with releases either from the Globe facility and/or from one or both of two other smelters which previously existed in the area (the Argo Smelter and the Omaha and Grant Smelter). The locations of these three smelters in relation to the VBI70 site are also shown in Figure ES-1.

Based on the results of several rounds of soil sampling, USEPA concluded that the VBI70 site contained multiple residences where the concentration of arsenic and/or lead in yard soil could be above a level of potential human health concern. On this basis, USEPA proposed the VBI70 site for inclusion on the Superfund National Priorities List (NPL) in January, 1999, and the site was added to the NPL on July 22, 1999.

2.0 SUMMARY OF SITE DATA AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN

2.1 Initial Studies

Phase I/Phase II

Once investigations at the nearby Globe site began to suggest that elevated levels of arsenic and/or lead might exist in soils at residential properties within the area of the VBI70 site, CDPHE requested assistance from USEPA Region VIII in characterizing the nature and extent of the contamination. In response, USEPA Region VIII undertook a study designed to identify

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properties that had levels of arsenic or lead that were sufficiently high that time-critical action (soil removal and replacement) might be warranted. Most of these samples were collected during the initial round of sampling (referred to as Phase I), with the remainder being obtained in a subsequent sampling effort (Phase II). In the majority of cases, two surface samples and one subsurface sample were collected per property, with additional surface samples at some locations (depending on the size of the property).

The action levels selected for time-critical soil removal were 450 ppm for arsenic and 2,000 ppm for lead. For arsenic, a majority of properties sampled (927 out of 1390) had maximum values that were below the limit of detection (average detection limit = 53 ppm). However, arsenic was detected in one or more surface soil samples at a number of properties, with 40 of these properties having one or more samples above 450 ppm. For lead, most properties (1,153 out of 1,390) had concentration values in surface soil that were below 400 ppm, but 238 properties had one or more values above 400 ppm. Of these, six properties have one or more lead value above 2,000 ppm.

In order to help confirm the identity of properties which warranted time-critical soil removal actions, USEPA collected two or more composite samples (each consisting of five sub-samples) of surface soil from residential properties where one or more grab samples were above the removal level for arsenic. Based on the results of this composite sampling program, a total of 21 residences were identified where one or more composites confirmed that arsenic levels were above the action level. Of these, 18 underwent soil removal and replacement in the fall of 1998, while the owners of the other three properties refused permission for the removal. No properties were identified where lead levels in composite soil samples were high enough to warrant a time-critical soil removal action.

Risk-Based Sampling Program

One of the striking findings that emerged from the Phase I/Phase II sampling programs was that arsenic-impacted properties did not appear to occur in a clear spatial pattern. That is, the occurrence of high arsenic levels in soil did not appear to be associated with proximity to one or more of the smelters, and properties with elevated levels of arsenic often occurred immediately adjacent to one or more residences that were not apparently impacted.

In order to obtain additional information on the spatial pattern of contamination both within and between yards, USEPA selected eight properties to undergo detailed soil sampling. Five of the yards were locations where Phase I/Phase II sampling indicated the arsenic concentrations were above the removal level, while three of the properties had arsenic concentrations below the removal level. At each property, a high-density grid was established on 5-foot centers, and soil samples were collected wherever the grid node did not fall on a driveway, patio, etc. In addition, whenever access could be obtained, the sampling grid was extended 10-15 feet into adjacent properties in order to determine if there was a clear difference in contamination levels between adjacent properties.

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The results for one property are shown in Figure ES-2. As seen at this location, there is a fairly clear boundary between the property of concern and the adjacent properties. Similar patterns are observed at other properties, although there are some locations where the contamination may extend somewhat into the adjacent property.

Other activities conducted under the Risk-Based Sampling Program included collection of a number of environmental samples (dust, water, paint, vegetables) at the eighteen properties selected for soil removal. Arsenic and lead levels in indoor dust were found to have no apparent relationship to levels in yard soil, suggesting that soil was not a predominant source of contaminant levels in indoor dust. Lead levels in tap water were all below the current USEPA action level for lead in drinking water (15 ug/L), suggesting that tap water is not likely to be a significant source of exposure. Lead was detected in paint at most locations, with 130 out of 144 samples having values above 1 mg/cm². These data suggest that interior and/or exterior leaded paint might be a source of lead exposure in area children, either directly (by paint chip ingestion), or indirectly (by ingestion of dust or soil containing paint chips). Only one of the 18 properties scheduled for soil removal had a vegetable garden. At this location, concentrations of arsenic and lead were below the level of detection in two vegetable samples. Because so few samples were obtained, no conclusions can be drawn from this data set.

In addition to environmental sampling, a number of biological samples (hair, urine, blood) were also collected from residents in the properties selected for soil removal. A total of 15 individuals residing at six of the properties scheduled for soil removal volunteered to participate in the program. None of the samples collected exceeded the normal range for lead or arsenic. Although this data set is too small to draw firm conclusions, the results suggest that exposures at these locations may not be of immediate health concern.

Physical-Chemical Characterization Study

USEPA also undertook a study to characterize the physical chemical attributes of the lead and arsenic contamination in residential site soils. Arsenic was found to occur mainly as arsenic trioxide, with a smaller fraction present as arsenic antimony oxide. Lead occurs in several phases, with the highest amount present as lead arsenic oxide. Other phases that are present include lead manganese oxide and lead phosphate, but these forms do not appear to increase as a function of total lead concentration, suggesting that these phases may be mainly natural in origin.

In addition, the concentration of metals in bulk (unsieved) soil samples were compared to that in fine (sieved) samples. For lead, cadmium, and zinc, the concentration measured in the fine fraction is, on average, very nearly equal to that in the bulk fraction, with the data lying very close to the line of identity. For arsenic, there is a tendency for the concentration values in the fine fraction to be somewhat higher than in the bulk fraction, with an average difference of about 20%.

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2.2 Selection of Chemicals of Potential Concern

Chemicals of potential concern (COPCs) were selected based on available data from full-suite analyses of soil samples for the 23 metals included on USEPA's Target Analyte List (TAL). In accord with standard methods identified in USEPA risk assessment guidance, chemicals were eliminated if: a) the maximum value was below a level of health concern, b) the chemical is a beneficial mineral that is required for good health, and c) if the risk contributed is minor compared to other chemicals that will be retained. Based on these selection procedures, the COPCs selected for quantitative evaluation at the VBI70 site are arsenic and lead. All other chemicals are either not of concern or are present at levels which contribute minimal risk compared to arsenic.

2.3 Phase III Investigation

Because of the absence of any clear spatial pattern of soil contamination, USEPA concluded that the identity and location of properties with elevated levels of arsenic and/or lead could not be reliably predicted using traditional approaches. For this reason, USEPA undertook a large-scale sampling program designed to obtain data that would help evaluate health risks to residents in the area. This program is referred to as the Phase III investigation. The investigation consisted of four main parts:

- Sampling of residential yard soils
- Sampling of indoor dust at residences
- Sampling of residential vegetable gardens (vegetables and soil)
- Supplemental sampling of soil at local schools and parks

Phase III was implemented in two parts. The first part, referred to as Phase IIIa, focused mainly on properties (including residences, schools, and parks) which had not been investigated in Phases I or II. The second part, referred to as Phase IIIb, consisted of re-sampling at properties that had previously been sampled in Phase I or II, but for which the data were judged to be too limited to support clear risk-management decision making.

At the time of the preparation of this public review draft, the results from the Phase IIIa effort are available, and are the basis for this risk assessment. The results from the Phase IIIb effort are not yet complete, but will be incorporated into the final risk assessment.

Residential Soil Sampling

A total of 30 surface soil (0-2 in.) grab samples were collected from each property where access was granted. These 30 samples were combined into three composite samples, each containing 10 grab samples. The composites were prepared by combining every third grab sample, such that each composite represents an independent estimate of the yard-wide mean concentration. All composite samples were dried and mixed, and then analyzed for arsenic and lead by XRF.

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The total number of properties targeted in Phase IIIa was about 2,600. Of these, a total of 1,637 granted USEPA authority to collect samples, and samples were successfully collected at a total of 1,548 properties. Summary statistics, based on average values at each property and stratified by neighborhood, are summarized in Table ES-1. For arsenic, most properties (1,270 out of 1,548) have average concentrations of 50 ppm or less, with 126 properties between 50-100 ppm, 106 between 100-200 ppm, and 46 above 200 ppm. For lead, 1389 properties have mean lead concentrations lower than 400 ppm, with 153 between 400-800 ppm and six higher than 800 ppm. There is only a weak correlation between the occurrence of elevated lead and elevated arsenic in soil, suggesting that the main sources of lead and the main sources of arsenic in yard soil are not likely to be the same.

Residential Dust Sampling

In accord with the initial results obtained during the Risk-Based sampling program, only a weak correlation was detected between the level of either arsenic or lead in paired soil and dust samples ($R^2 = 0.14$ to 0.18 , respectively). Nevertheless, the slopes of both regression lines are statistically different from zero ($p < 0.01$), with best estimate parameter values as follows:

$$\text{Arsenic:} \quad C_{\text{dust}} = 0.06 \cdot C_{\text{soil}} + 11$$

$$\text{Lead:} \quad C_{\text{dust}} = 0.34 \cdot C_{\text{soil}} + 150$$

Residential Garden Sampling

USEPA collected 72 samples of different types of garden vegetables from 19 different properties around the site. At each location, samples of garden soil were also collected. For arsenic, the concentrations in garden soils were generally lower than in the corresponding yard soils, with no significant relationship between the two. For lead, garden soils also tended to be lower than for yard soils, but there was a weak correlation between the two. These data suggest that garden soils are not equivalent to yard soils, presumably because most gardens are amended by addition of soil, peat, fertilizer, etc. The mean concentrations of arsenic and lead in garden vegetables were 0.044 and 0.15 ug/g wet weight (ww), respectively. There was no apparent difference in concentration as a function of vegetable type (exposed, protected, root). There is very little tendency for the concentration of either lead or arsenic in garden vegetables to increase as a function of the concentration of in garden soil, although there is a slight (and statistically significant) trend for arsenic (slope = 0.002 mg/kg ww per mg/kg in soil). The slope for lead is very close to zero and is not statistically significant.

Sampling at Schools and Parks

Samples of surface soil were collected at 10 schools and one park. Concentrations of arsenic are generally low, with average values ranging from 11-15 ppm, and maximum values less than 30 ppm. An exception to this pattern occurred at one school property where two values significantly

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higher than expected were detected (1,517 ppm and 70 ppm). These values occur adjacent to each other, and are surrounded by values of 17-23 ppm. This suggests there might be a small "hot spot" at this location.

2.4 Data Selected For Use in This Risk Assessment

The data from the Phase IIIa sampling program were selected for use in this risk assessment because 1) all Phase IIIa data were collected in accordance with project plans that were developed with careful consideration of the Data Quality Objectives (DQOs) needed to support risk assessment calculations, and 2) all data collected during Phase IIIa are accompanied by thorough Quality Assurance (QA) data that allow detailed evaluation of the reliability of the data. A detailed review of these quality assurance data reveal that the data collected are of high quality, with adequate accuracy and precision to support a reliable evaluation of human health risk.

Data collected during Phase I/Phase II were not used because they were collected only with the intent of identifying locations that exceeded the removal action levels, and were not intended to support risk calculations or remedial decision making. More specifically, data from Phase I/Phase II were not used because 1) many samples had elevated detection limits for arsenic, 2) the sampling density at each property was too low, and/or 3) sampling locations were not clear. However, despite these limitations, it is clear that the data from Phase I/Phase II and from Phase IIIa are generally similar, each indicating the occurrence of scattered properties with elevated levels of lead and/or arsenic.

3.0 EXPOSURE ASSESSMENT

Figure ES-3 presents a conceptual model showing the main pathways by which contaminants present in surface soil may come into contact with area residents. Exposure scenarios that are considered most likely to be of concern are shown by boxes containing a solid circle, and greatest attention is focused on these pathways. Pathways which are judged to contribute only occasional and minor exposures are shown by boxes with an open circle. Incomplete pathways (i.e., those which are not thought to occur) are shown by open boxes. Based on this conceptual model, the following pathways are judged to be of sufficient potential concern to warrant quantitative exposure and risk analysis:

Population	Medium and Exposure Route
Resident	Incidental ingestion of soil and dust in and about the home and yard
	Ingestion of home-grown vegetables

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Other exposure pathways are judged to be sufficiently minor that further quantitative evaluation is not warranted.

4.0 QUANTIFICATION OF EXPOSURE AND RISK FROM ARSENIC

4.1 Quantification of Exposure

Exposure of residents to arsenic in soil, dust and vegetables were evaluated using the standard exposure equations identified in USEPA risk assessment guidance. Exposure parameter input values were the default values recommended in USEPA guidance for residents. All concentration values in soil, dust and garden vegetables were based on site-specific data.

4.2 Toxicity Assessment

The toxic effects of arsenic have been reasonably well established, based mainly on studies of humans exposed to elevated levels of arsenic from a variety of sources. The findings from these studies are summarized briefly below.

Noncarcinogenic Effects

Oral exposure to high doses of arsenic produces marked irritation of the gastrointestinal tract, leading to nausea and vomiting. Symptoms resulting from chronic ingestion of lower doses of arsenic often begin with a vague weakness and nausea. As exposure continues, symptoms become more characteristic and may include signs such as diarrhea, vomiting, anemia, injury to blood vessels, damage to kidney and liver, and impaired nerve function that leads to "pins and needles" sensations in the hands and feet. The most diagnostic sign of chronic arsenic exposure is an unusual pattern of skin abnormalities, including dark and white spots and a pattern of small "corns," especially on the palms and soles.

Carcinogenic Effects

There is strong evidence from a number of human studies that oral exposure to arsenic increases the risk of skin cancer. The most common type of cancer is squamous cell carcinoma, which appears to develop from some skin corns. In addition, basal cell carcinoma may also occur, typically arising from cells not associated with the corns. Although these cancers may be easily removed, they can be painful and disfiguring and can be fatal if left untreated. More recent data indicate that chronic oral arsenic exposure may also increase the risk of internal cancers, including cancer of the bladder and lung.

Toxicity Factors for Arsenic

Based on the available toxicity data for arsenic, the USEPA has established both a Reference Dose (RfD) for evaluating risk of non-cancer effects, and a cancer slope factor for quantifying

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the risk of cancer. These values are summarized below.

Toxicity Factor	Value	Source
Chronic RfD	0.0003 mg/kg-day	IRIS 2000
Oral Slope Factor	1.5 (mg/kg-day) ⁻¹	IRIS 2000

Because the oral RfD and the oral SF for arsenic are based on studies of humans exposed to arsenic either in drinking water or in other readily absorbable forms, solid forms of arsenic in site soils may be less well-absorbed and require adjustments in the toxicity factors to derive appropriate estimates of toxicity. In order to investigate the relative bioavailability (RBA) of arsenic in site soils, USEPA performed a study in which five separate samples were fed to swine for 12 days. The study found that arsenic in site soils was less well absorbed than a readily soluble form of arsenic (sodium arsenate), with RBA values for individual samples ranging from about 0.2 to 0.5. Based on the results of this study, the upper confidence limit of the RBA for arsenic in site soils was estimated to be 0.5.

4.4 Risk Characterization for Arsenic

Risks from Soil and Dust

Cancer Risk

Cancer risks from exposure of residents to arsenic in yard soil and indoor house dust were calculated for each property using the basic equations recommended by USEPA. The resulting risk estimates are shown in Table ES-2.

For central tendency exposure (CTE) conditions, most properties have estimated excess cancer risks for exposures due to arsenic in soil plus dust that lie between 1.1E-06 and 1.4E-05 (5th to 95th percentile), with a maximum value of 8.3E-05.

For reasonable maximum exposure (RME) conditions, most properties have risk estimates that lie between 1.0E-05 and 1.3E-04, with a maximum value of 7.8E-04. Of the total properties investigated (1,548), 112 (approximately 7%) have RME cancer risks which exceed 1.0E-04. The spatial pattern of properties with arsenic contamination that exceeds a risk level of 1E-04 is approximately uniform across the site, with an average frequency of about 8%.

Chronic Noncancer Risks

Estimated risks of non-cancer health effects from chronic exposure to arsenic in soil and dust are shown in Table ES-3. For individuals with CTE, risks at most properties fall between 2E-02 and 2E-01 (5th to 95th percentile), while individuals with RME have risks that lie mainly between

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5E-02 and 7E-01. These results indicate that risk of noncancer effects is low for most individuals at most locations. However, there is one location where the CTE non-cancer risk does slightly exceed 1E+00 (HQ = 1.4E+00), and there are 28 locations where the RME hazard quotient (HQ) values exceed 1E+00 (maximum = 4E+00). These locations where noncancer risks enter a range of concern (HQ > 1E+00) are also above the usual level of concern (1E-04) for cancer.

Noncancer Risks from Short-Term Exposures

In most cases, if chronic noncancer and cancer risks from arsenic are below a level of concern, risks from shorter term exposures will also be below a level of concern. However, in cases where there is high spatial variability of arsenic concentrations in soil at different locations within a property (as may occur at this site), it is conceivable that long-term average exposures in a yard might not be of concern, but that short-term high-intake exposures by a child playing in the yard at locations of above-average concentration could be of concern. This is commonly referred to as a "hot-spot" exposure scenario.

The USEPA has not established standard exposure parameters for evaluating short-term exposures, so screening level calculations were performed based on the following assumptions:

Concentration = maximum value that could be contained in any composite value
Body weight = 10 kg (this corresponds to a child who is 6-12 months in age)
Soil Intake rate = 500 mg/day (sub-chronic) or 2,000 mg/day (sub-acute)
Exposure Frequency = 30/120 (sub-chronic) or 1/2 (sub-acute)
RfD = 6E-03 mg/kg-day (sub-chronic) or 5E-02 mg/kg-day (sub-acute)

Based on the exposure assumptions above, the following results are obtained:

Statistic	Sub-Acute	Sub-Chronic
N < 1E+00 (no concern over hot spots)	1387	1382
N > 1E+00 (possible hot spot)	161	166
N > 1E+00 AND not of chronic concern (risk < 1E-04)	49	54

As seen, about 161-166 properties are identified by this screening technique as locations where a hot-spot of potential short-term noncancer health concern might exist. Of these properties, most are already identified as being of potential concern due to estimated chronic cancer risks which exceed a cancer risk level of 1E-04. The remaining properties (about 49-54) are locations where additional investigation may be needed to determine if any short-term risks are actually present.

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Risks from Home-Grown Vegetables

A total of 72 different samples of garden vegetables were collected from 19 different properties across the site. At each property, the 95% upper confidence limit (UCL) of the mean concentration of arsenic was calculated, and this value (or the maximum, whichever was lower) was used to estimate risks to residents. For individuals whose intake of home-grown garden vegetables is average (CTE) for the western United States, neither non-cancer nor cancer risks enter a range of concern at any property tested. For individuals whose intake is at the upper-bound (RME) of the distribution of garden vegetable consumption, risks do enter a range of potential concern for five properties. At three of these properties, the magnitude of the excess risk is relatively small (RME cancer risk = $2E-04$), and is due in most cases to the conservatism introduced by use of the 95% UCL of the mean rather than the mean concentration for the risk calculations. At the fourth property, the high risk estimates are attributable to a single vegetable sample (garlic) that was significantly higher than the remainder of the samples from this location. Analysis of a second garlic sample from this location yielded a much lower arsenic concentration, suggesting that the original value may have been biased high. At the fifth property, a number of vegetables had concentration values that were higher than in samples from most other properties, and samples from this location could be of potential concern for an RME consumer. The concentrations of arsenic in the garden soil samples at this location were also somewhat higher (mean = 51 ppm) than for most other gardens (average = 15 ppm, range = 11 to 24 ppm), suggesting the elevated values were attributable to soil contamination. An interview with the property owner did not reveal any probable source of arsenic in the garden.

4.5 Uncertainties in Arsenic Risk Assessment

It is important to recognize that the calculations of short-term and long term exposure and risk from arsenic ingestion in soil are based on a number of assumptions and estimates, and that these introduce uncertainty into the risk results. The most important of the sources of uncertainty in the calculations are summarized below.

Uncertainty in Concentration Terms

The concentration term that is appropriate for calculating exposure and risk from ingestion exposure to arsenic is the true mean concentration in the medium of concern (soil, dust, vegetables), averaged over the area and time interval (averaging time) of concern. Because the true mean cannot be calculated from a limited set of sample results, the USEPA utilizes the 95% upper confidence limit of the mean as a conservative estimate of the true mean. This approach helps ensure that the exposure and risk estimates that are derived are more likely to overestimate than underestimate the actual risk.

Uncertainty in Intake Rates

Data on the amount of soil ingested by humans are very limited. Measurements are difficult to perform, and results vary significantly from study to study and from method to method. In

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addition, data are based mainly on short term studies, so estimates of long-term average intake rates are especially uncertain. Moreover, intake rates are likely to vary from site to site and property to property, depending on things such as climate, socioeconomic status, yard condition, etc, so the default intake rates used in these calculations may not reflect the true intake rates at the site. Because of the limitations in the data, the default values recommended by USEPA are intended to be on the high side (i.e., are more likely to overestimate than underestimate actual soil ingestion).

Uncertainty in Exposure Duration

Cancer risk calculations depend on the duration of exposure. Default exposure durations used in the risk assessment are not site-specific, and are estimated from data on the length of time that people own a particular residence. Thus, actual exposure durations of residents at the site may not be the same as the assumed exposure durations assumed, and might be either longer or shorter than assumed.

Uncertainty in Toxicity Factors

One of the largest sources of uncertainty in most risk assessments stems from uncertainty in the toxicity factors used to predict responses from the calculated doses. In the case of arsenic, dose-response data are derived from studies in humans, which significantly reduces the degree of uncertainty compared to extrapolations based on animal data. However, a significant degree of uncertainty still remains in all of the toxicity factors. For example, dose-response curves in the key studies are generally limited by lack of precise data on the actual exposure rates. Moreover, there are still large uncertainties in how to extrapolate from relatively high exposure levels to lower exposure levels, and in the importance of cultural and ethnic differences between different study populations. USEPA is currently in the process of reassessing the risk characterization for arsenic, and the quantitative risk factors (RfD and/or oral slope factor) may be revised in the future as new data and as new analyses warrant.

Uncertainty in Bioavailability

In order to cause an adverse response, arsenic that is ingested must be absorbed into the body. Measurements of the arsenic relative bioavailability have been performed for five soils from the VBI70 site. While measurements based on site soils significantly reduces uncertainty in this exposure parameter, uncertainty still remains. For example, variability was observed between different site soils, and a conservative estimate of the mean value was employed to represent the site-wide average absorption. This approach is expected to result in an over-estimate of true absorption. Another source of uncertainty is in the extrapolation of data from test animals to humans. The test animals (swine) were selected because they are believed to have a gastrointestinal system similar to that in humans, but it is also expected that absorption in humans may vary as a function of age, stomach contents, nutritional status, etc. Thus, the measurements in animals should be viewed as uncertain estimates of the true values in humans.

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Summary

Because of the uncertainties summarized above, none of the exposure and risk calculations for arsenic should be interpreted as accurate measures of the true risk, rather, all values should be interpreted as uncertain estimates. Because most of the approaches for dealing with uncertainty are intended to be conservative (i.e., are more likely to overestimate than underestimate), the risk values above should be thought of as high-end estimates of the true risk, and actual risks are likely to be lower.

5.0 EXPOSURE AND RISK FROM LEAD

5.1 Overview

Risks from lead are evaluated using a somewhat different approach than for most other metals. First, risks are assessed only for young children because they are more susceptible to lead than adults. Second, risks are expressed at the probability that a child will have a blood lead value greater than 10 ug/dL. The health-based goal established by USEPA is that there should be no more than a 5% chance that any child will have a blood lead value above 10 ug/dL.

5.2 IEUBK Model for Assessing Lead Risk

Risks from Soil and Dust

The USEPA has developed an Integrated Exposure Uptake Biokinetic (IEUBK) model for predicting the likely range of blood lead levels in a population of young children (age 0-6 years) exposed to a specified set of environmental lead levels. Based in part on this model, the USEPA has established a national policy that soil lead levels below 400 ppm may be assumed to be below a level of health concern. Soil lead levels above the 400 ppm screening level may or may not be of concern, depending on site-specific factors.

The IEUBK model was used to predict risks at each property that was sampled during Phase IIIa, using default IEUBK model input parameters for all parameters except for the concentration of lead in soil and dust, which were based on site-specific measurements. The results are shown in Table ES-4. As seen, of the total of 1,548 residences examined, a total of 159 (10%) have mean soil lead levels above EPA's screening level of 400 ppm. In most of these cases the soil lead is only slightly elevated, with 137 of the 159 being less than 600 ppm. When characterized in terms of predicted risk of exceeding a blood lead level of 10 ug/dL (this is referred to as "P10"), the majority of homes (108 out of 159) above the 400 ppm soil lead level would be expected to have P10 values of 5-10%, only slightly above the health-based goal of 5%. However, about 40 properties would be expected to have P10 values between 10-20%, and 11 homes are predicted to have P10 values greater than 20%.

Although homes with elevated soil lead are found in all neighborhoods, the density of homes above 400 ppm tends to be higher in the central and western part of the site than in areas on the

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eastern side of the site.

Risks from Lead in Garden Vegetables

As noted previously, there is essentially no uptake of lead from soil into garden vegetables at this site. On this basis, it is concluded that exposure to lead from ingestion of home grown garden vegetables is not of concern.

5.3 Uncertainties in Lead Risk Evaluation

It is important to stress that lead risk predictions based on the IEUBK model are uncertain. This uncertainty arises from a number of factors. First, there is inherent difficulty in providing the model with reliable estimates of human exposure to lead-contaminated media. For example, exposure to soil and dust is difficult to quantify because human intake of these media is likely to be highly variable, and it is very difficult to derive accurate measurements of actual intake rates. Likewise, site-specific data on exposure to lead through the diet are generally not available, and because dietary lead levels have been decreasing over time, the default data used in the model may no longer be accurate. Second, it is often difficult to obtain reliable estimates of key pharmacokinetic parameters in humans (e.g., absorption fraction, distribution and clearance rates, etc.), since direct observations in humans are limited. Finally, the absorption, distribution and clearance of lead in the human body is an extremely complicated process, and any mathematical model intended to simulate the actual processes is likely to be an over-simplification. Consequently, IEUBK model calculations and predictions should not be thought of as being identical to actual risk.

One way to help determine whether the IEUBK model is yielding reliable results at a particular site is to compare the IEUBK model predictions with actual observations of blood lead levels in the population of children currently living at the site. At the VBI70 site, only very limited blood lead data are available, with values from only 15 individuals available. In this group of individuals, no cases of blood lead values above 10 ug/dL were observed. However, the data set is too limited to support any strong conclusion.

Another way to evaluate the potential uncertainties that may be associated with the use of the IEUBK model in evaluating lead risks to children is to consider the results of alternative approaches. For example, USEPA Region VIII has been working to develop a variation of the IEUBK model that is referred to as the Integrated Stochastic Exposure (ISE) Model for Lead. This model is similar to the IEUBK model, except that it uses probability density functions (PDFs) rather than point estimates as inputs for most concentration and exposure parameters. These distributions are combined using Monte Carlo simulation techniques to yield a predicted distribution of absorbed lead doses (ug/day) for different members of the exposed population. These doses are then used as input to the biokinetic portion of the IEUBK model in order to generate the predicted distribution of blood lead values in the population. Thus, the variability

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between children is evaluated in the ISE model based on the variability in environmental and exposure parameters, rather than by application of an assumed or estimated GSD value as in the IEUBK model. When the ISE model is used to estimate the relation between soil lead and blood lead at the VBI70 site, lead risks from soil are not as large as predicted by the IEUBK model, and most properties within the site would be of low health concern. The difference in the conclusions between the IEUBK model and the ISE model illustrate the range of uncertainty which is associated with estimating risks to children from lead in soil.

6.0 CONCLUSION

Arsenic occurs in soil at some residential properties at the VBI70 site at concentration levels that pose an RME excess lifetime cancer risk above a level of $1E-04$. Based on current data, about 8% of all properties fall into this category. Non-cancer risks from arsenic are also above a level of human health concern at some properties, mainly at the same locations where cancer risks are of concern. However, non-cancer risks from short-term exposures to arsenic at small "hot spots" may occur at some locations where cancer risks are below a level of concern. EPA will perform further sampling at these locations to determine if short-term exposures are actually of concern or not.

Properties with elevated levels of arsenic occur at widely scattered locations across the site, with no clear spatial pattern. At an impacted property, the contamination appears to be distributed across the yard area, with a fairly clear boundary between the impacted property and the adjacent properties. The chemical form of the arsenic is predominantly arsenic trioxide.

Lead also occurs at elevated levels in soil at some residential properties. Using EPA's IEUBK model to evaluate the risk to children, it is estimated that about 10% of residences have levels that exceed EPA's health-based goal (no more than a 5% chance that a child will have a blood lead value above 10 ug/dL). Of these 10%, about 7% have only slightly elevated lead levels and exceed the health-based goal by only a small amount, while about 3% of the properties have lead levels that are substantially above the target risk level. The pattern of properties with lead contamination does not appear to be closely linked to those that are impacted by arsenic.

TABLE ES-1 SUMMARY STATISTICS FOR PHASE IIIa SOIL SAMPLES

ARSENIC

Statistic	Clayton	Cole	Five Points	Globeville	Swansea/Elyria	All
N	644	601	14	22	267	1548
5th	5.5	5.5	5.5	5.5	5.5	5.5
25th	5.5	7.7	10.3	8.3	5.5	5.5
50th	10.3	11.8	16.4	10.8	9.0	10.9
75th	40.5	23.9	21.9	18.3	23.9	30.7
90th	109.7	91.4	28.4	33.6	93.1	98.4
95th	175.9	150.7	39.7	44.8	140.3	161.9
Max	609.6	659.8	59.2	70.9	430.6	659.8

LEAD

Statistic	Clayton	Cole	Five Points	Globeville	Swansea/Elyria	All
N	644	601	14	22	267	1548
5th	79	135	170	170	82	87
25th	111	216	383	226	119	135
50th	147	284	436	282	177	204
75th	201	367	549	331	281	309
90th	286	452	687	383	409	402
95th	348	515	764	424	479	475
Max	1131	1004	835	782	922	1131

All values are based on the mean value observed at each property

TABLE ES-2 ESTIMATED CANCER RISK FROM ARSENIC IN SOIL AND DUST

Neighborhood	Properties Evaluated	CTE Cancer Risk				RME Cancer Risk			
		≤ 1E-05	1E-05 - 1E-04	1E-04 - 1E-03	> 1E-03	≤ 1E-05	1E-05 - 1E-04	1E-04 - 1E-03	> 1E-03
Clayton	644	587	57	0	0	219	371	54	0
		91.1%	8.9%	0.0%	0.0%	34.0%	57.6%	8.4%	0.0%
Cole	601	554	47	0	0	124	437	40	0
		92.2%	7.8%	0.0%	0.0%	20.6%	72.7%	6.7%	0.0%
Five Points	14	14	0	0	0	2	12	0	0
		100.0%	0.0%	0.0%	0.0%	14.3%	85.7%	0.0%	0.0%
Globeville	22	22	0	0	0	4	18	0	0
		100.0%	0.0%	0.0%	0.0%	18.2%	81.8%	0.0%	0.0%
Swansea/Elyria	267	247	20	0	0	92	157	18	0
		92.5%	7.5%	0.0%	0.0%	34.5%	58.8%	6.7%	0.0%
All Neighborhoods	1548	1424	124	0	0	441	995	112	0
		92.0%	8.0%	0.0%	0.0%	28.5%	64.3%	7.2%	0.0%

TABLE ES-3 ESTIMATED CHRONIC NONCANCER RISK FROM ARSENIC IN SOIL AND DUST

Neighborhood	Properties Evaluated	CTE Hazard Quotient					RME Hazard Quotient				
		≤1	1-2	2-5	6-10	> 10	≤1	1-2	2-5	6-10	> 10
Clayton	644	643	1	0	0	0	633	7	4	0	0
		99.8%	0.2%	0.0%	0.0%	0.0%	98.3%	1.1%	0.6%	0.0%	0.0%
Cole	601	601	0	0	0	0	585	10	6	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	97.3%	1.7%	1.0%	0.0%	0.0%
Five Points	14	14	0	0	0	0	14	0	0	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	100.0%	0.0%	0.0%	0.0%	0.0%
Globeville	22	22	0	0	0	0	22	0	0	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	100.0%	0.0%	0.0%	0.0%	0.0%
Swansea/Elyria	267	267	0	0	0	0	266	1	0	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	99.6%	0.4%	0.0%	0.0%	0.0%
All Neighborhoods	1548	1547	1	0	0	0	1520	18	10	0	0
		99.9%	0.1%	0.0%	0.0%	0.0%	98.2%	1.2%	0.6%	0.0%	0.0%

TABLE ES-4 ESTIMATED RISKS TO CHILDREN FROM LEAD IN SOIL AND DUST

Neighborhood	Count	Count >400	P10 >=5 and <10	P10 >=10 and <20	P10 >=20
CLAYTON	644	18	11	4	3
	100%	2.8%	1.7%	0.6%	0.5%
COLE	601	104	77	23	4
	100%	17.3%	12.8%	3.8%	0.7%
FIVE POINTS	14	7	3	3	1
	100%	50.0%	21.4%	21.4%	7.1%
GLOBEVILLE	22	2	1	0	1
	100%	9.1%	4.5%	0.0%	4.5%
SWANSEA/ELYRIA	267	28	16	10	2
	100%	10.5%	6.0%	3.7%	0.7%
ALL	1548	159	108	40	11

FIGURE ES-2 SPATIAL DISTRIBUTION OF CONTAMINANTS -- PROPERTY 1

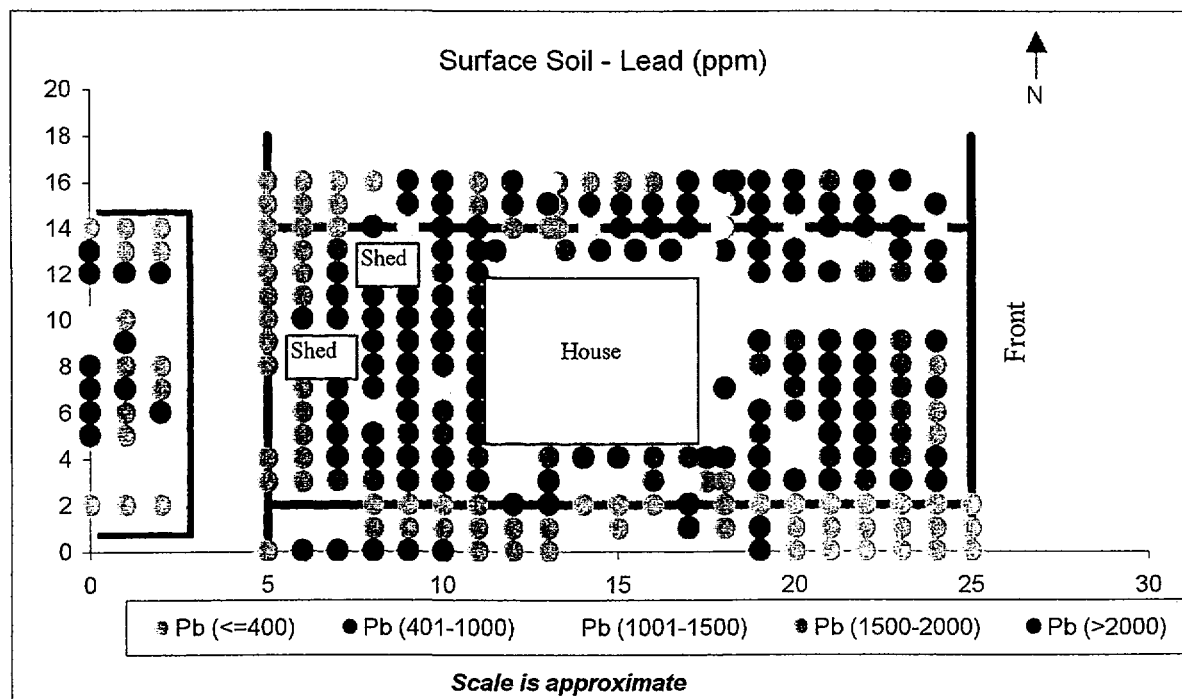
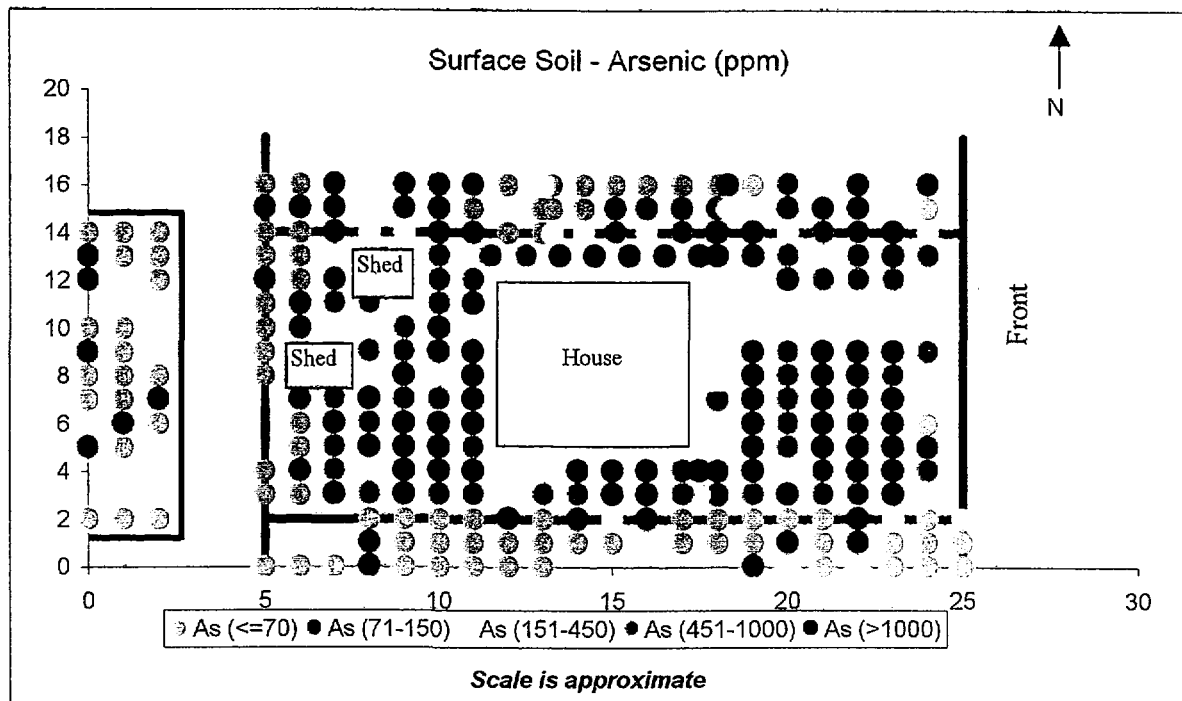
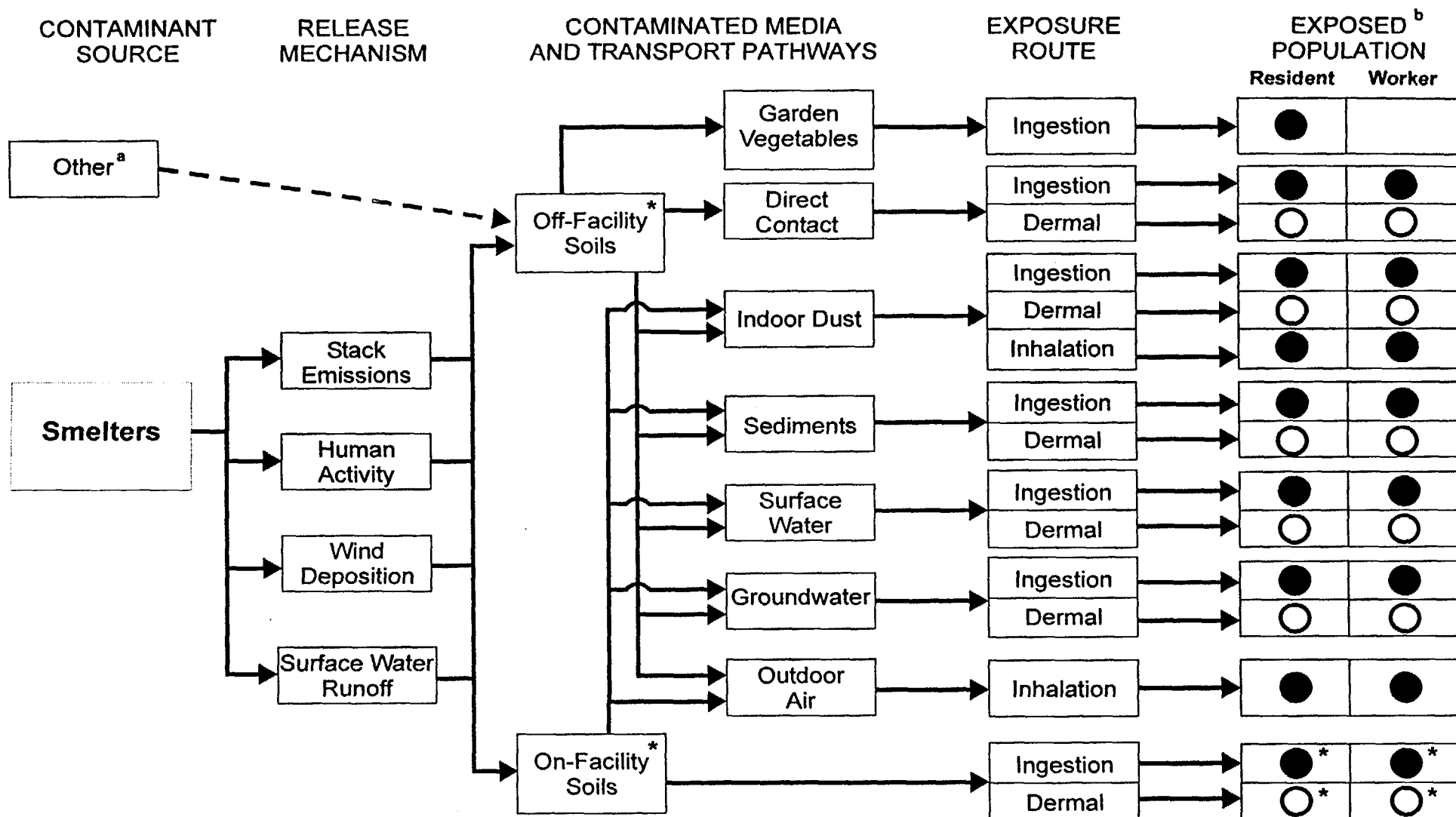





FIGURE ES-3 CONCEPTUAL SITE MODEL
POTENTIAL HUMAN EXPOSURE PATHWAYS AT VASQUEZ BLVD./I-70 SITE



-  = Pathway is not complete
-  = Pathway is complete, but minor; qualitative evaluation
-  = Pathway is complete and could be significant; quantitative evaluation

* "On-Facility" exposure is only at the former Omaha-Grant and Argo sites.

^a - Other sources may be historical smelters, other active smelters & arsenical pesticides.

^b - The work group will refine the list of exposed populations as the risk assessment proceeds and

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List of Acronyms and Abbreviations

CDC	Centers for Disease Control
CDPHE	Colorado Department of Public Health and Environment
COPC	chemical of potential concern
CTE	central tendency exposure
DI	daily intake
DL	detection limit
dw	dry weight
EC01	concentration in water that results in a 1% increase in excess lifetime cancer risk
EMPA	electron microprobe analysis
EPC	exposure point concentration
GFAA	graphite furnace atomic absorption
GSD	geometric standard deviation
HIF	human intake factor
HQ	hazard quotient
ICP	inductively coupled plasma spectroscopy
MS	mass spectrometry
IEUBK	Integrated Exposure Uptake Biokinetic
IRIS	Integrated Risk Information System
ISE	Integrated Stochastic Exposure
LOAEL	lowest observed adverse effect level
MTHC	maximum theoretical hotspot concentration
NOAEL	no observed adverse effect level
NPL	National Priorities List
PbB	blood lead level
PDF	probability density function
ppm	parts per million
RBA	relative bioavailability
RBC	risk-based concentration
RfD	reference dose
RME	reasonable maximum exposure
SF	slope factor
TAL	Target Analyte List
UCL	upper confidence limit
USEPA	U.S. Environmental Protection Agency
VBI70	Vasquez Boulevard and I-70 Site
ww	wet weight
XRF	X-ray fluorescence

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SECTION 1 INTRODUCTION

1.1 SITE DESCRIPTION

The Vasquez Boulevard and I-70 (VBI70) Superfund Site is an area of approximately four square miles located in the north-central section of Denver, Colorado. The site is composed of a number of neighborhoods that are largely residential, including Swansea/Elyria, Clayton, Cole, and portions of Globeville. Most residences at the site are single family dwellings, but there are also some multi-family homes and apartment buildings. The site also contains a number of schools, parks, and playgrounds, as well as a number of commercial and industrial properties. Figure 1-1 is a map which displays the site.

The site is largely flat in topography, sloping gently towards the Platte River which flows in a northeasterly direction through the site. Other than the Platte River, there are no other major surface water bodies within the site.

The climate of the site is generally typical of Colorado's semiarid eastern plains. Temperatures are moderate throughout the year, with monthly averages ranging from 30° F in January to 73° F in July. Annual rainfall measures 16 inches, 60% of which falls during the spring and summer. The rainiest month is May, with an average rainfall of 2.6 inches. Snowfall totals in the Denver Metro area average 60 inches, with March usually receiving the most snow (12.5 inches). The Rocky Mountain foothills, about 20 miles west of the site, help create a predominantly southern wind flow at the site, with an annual average velocity of about 8.5 mph. Peak winds can reach velocities of 30-50 mph, with the highest winds tending to be from the north-northwest (Colorado Climate Center 2000).

1.2 BASIS FOR POTENTIAL CONCERN

The site came to the attention of the U.S. Environmental Protection Agency (USEPA) because studies directed by the Colorado Department of Public Health and Environment (CDPHE) at a nearby site (Globe Smelter) indicated that elevated concentrations of arsenic and/or lead occurred in the soil of some residential properties in the Swansea/Elyria area. The source of these elevated levels is not known, but *a priori*, it is considered plausible that the contamination is associated with releases either from the Globe facility and/or from one or both of two other smelters which previously existed in the area (the Argo Smelter and the Omaha and Grant Smelter). The locations of these three smelters in relation to the VBI70 site are also shown in Figure 1-1.

Based on the results of several rounds of soil sampling (see Section 2.0), USEPA concluded that the VBI70 site contained multiple residences where the concentration of arsenic and/or lead in

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yard soil could be above a level of potential human health concern. On this basis, USEPA proposed the VBI70 site for inclusion on the Superfund National Priorities List (NPL) in January, 1999, and the site was added to the NPL on July 22, 1999.

1.3 PURPOSE AND SCOPE OF THIS DOCUMENT

This document is a baseline human health risk assessment. The purpose of the assessment is to characterize the nature and magnitude of any risk to humans that may be attributable to contamination of site media, assuming that no steps are taken to remediate the environment or to reduce human contact with contaminated environmental media. More specifically, this assessment focuses on the direct and indirect risks to humans from contamination that is present in soils in current residential and commercial (non-smelter) areas of the site. This is referred to as the "Off Smelter Facility Operable Unit". The potential human health risks from exposure to other potentially contaminated environmental media (e.g., surface water, groundwater) will be investigated and evaluated as a separate operable unit.

The results of this baseline risk assessment are intended to help inform risk managers and the public about the level of health risk which is attributable to contamination in site soils, to help determine the need for remedial action at the site, and to provide a basis for determining the levels of chemicals that can remain in site soils and still be adequately protective of public health (USEPA 1989).

The methods used to evaluate risks to humans and the environment employed in this assessment are consistent with current guidelines provided by the USEPA for use at Superfund sites (USEPA 1989, 1991a, 1991b, 1991c, 1992a, 1992b, 1993a).

1.4 ORGANIZATION OF THIS DOCUMENT

In addition to this introduction, this report is organized into the following sections:

- | | |
|-----------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Section 2 | This section provides a summary of the available data on the levels of smelter-related chemicals (metals) in site soils, and identifies which of these chemicals are of potential health concern to area residents. |
| Section 3 | This section discusses how residents and other people (workers, children at schools or playgrounds) may be exposed to site-related chemicals, now or in the future, and identifies exposure scenarios that are considered to be of potential concern. |
| Section 4 | This section assesses the level of exposure and risk to humans from arsenic in site soils. This includes 1) a description of methods used to quantify exposure to arsenic, 2) data on the toxicity of arsenic to humans, 3) calculation of the level of noncancer and cancer risk that may occur as a result of exposure to arsenic in site |

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soils, and 4) a discussion of the uncertainties which limit confidence in the assessment.

- Section 5 This section assesses the level of exposure and risk to area residents from lead in site soils. This includes 1) a description of the toxic effects of lead, 2) a summary of the method used by USEPA to evaluate risks from lead, 3) a summary of the estimated risks at this site attributable to lead in site soils, and 4) a discussion of the uncertainties which limit confidence in the assessment.
- Section 6 This section provides full citations for USEPA guidance documents, site-specific studies, and scientific publications referenced in the risk assessment.

TARGET SHEET
EPA REGION VIII
SUPERFUND DOCUMENT MANAGEMENT SYSTEM

DOCUMENT NUMBER: 489833

SITE NAME: VASQUEZ BLVD. / I-70
DOCUMENT DATE: 01-JUL-00

DOCUMENT NOT SCANNED

Due to one of the following categories:

- ☐ PHOTOGRAPHS
- ☐ 3 - DIMENSIONAL
- ☐ OVERSIZED
- ☐ AUDIO/ VISUAL
- ☐ PERMANENTLY BOUND DOCUMENTS
- ☐ POOR LEGIBILITY
- ☒ OTHER COLOR MAP
- ☐ NOT AVAILABLE

FIGURE ES-1
SITE MAP

☐ TYPES OF DOCUMENTS NOT TO BE SCANNED
(Data Packages, Data Validation, Sampling Data, CBI, Chain of Custody)

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(Data Packages, Data Validation, Sampling Data, CBI, Chain of Custody)

FIGURE 1-1
SITE MAP

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SECTION 2 SUMMARY OF SITE DATA AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN

Data on the level of arsenic, lead, and other metals which might have been released from area smelters into site soils have been collected in a phased series of investigations. Each of these phases is described below, along with a summary of the key data collected during each phase.

2.1 PHASE I/PHASE II GRAB SAMPLE INVESTIGATION

Residential Soil Samples

Once investigations at the nearby Globe site began to suggest that elevated levels of arsenic and/or lead might exist in soils at residential properties within the area of the VBI70 site, CDPHE requested assistance from USEPA Region VIII in characterizing the nature and extent of the contamination. In response, USEPA Region VIII undertook a study designed to identify properties that had levels of arsenic or lead that were sufficiently high that time-critical action (soil removal and replacement) might be warranted. The action levels selected for time-critical soil removal were 450 parts per million (ppm) for arsenic and 2,000 ppm for lead (USEPA 1998a).

Details of the study are presented in UOS (1998a, 1998b). In brief, grab samples of surface soil and subsurface soil were collected from 1390 residential properties in the area of potential concern. Most of these samples were collected during the initial round of sampling (referred to as Phase I), with the remainder being obtained in a subsequent sampling effort (Phase II). In the majority of cases, two surface samples and one subsurface sample were collected per property, with additional surface samples at some locations (depending on the size of the property). All samples were analyzed for arsenic, lead, cadmium and zinc using X-ray fluorescence (XRF).

The results for arsenic in surface soil are summarized in Figure 2-1 (upper panel). As seen, a majority of properties sampled (927 out of 1390) had maximum arsenic values that were below the limit of detection (average detection limit = 53 ppm). However, arsenic was detected in one or more surface soil samples at a number of properties, with 40 of these properties having one or more samples above 450 ppm. Arsenic concentrations in subsurface samples were generally somewhat lower than the concentrations in surface soil, with an average ratio of subsurface to surface soil of about 0.8.

For lead (lower panel), most properties (1153 out of 1390) had maximum concentration values in surface soil that were below 400 ppm, but 238 properties had one or more values above 400 ppm. Of these, 6 properties had one or more lead values above 2,000 ppm. Lead levels in subsurface soil

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tended to be lower than in surface soil, with an average ratio of subsurface to surface soil of about 0.7.

Any property with one or more arsenic values above 450 ppm and/or one or more lead values above 2,000 ppm were identified as candidates for soil removal, pending verification by collection and analysis of composite soil samples (see below).

2.2 PHASE II CONFIRMATORY SAMPLING AND SOIL REMOVAL

In order to help confirm the identity of properties which warranted time-critical soil removal actions, USEPA collected two or more composite samples (each consisting of five sub-samples) of surface soil from residential properties where one or more grab samples were above the removal level for arsenic.

Based on the results of this composite sampling program, a total of 21 residences were identified where one or more composites confirmed that arsenic levels were above the action level. Of these, 18 underwent soil removal and replacement in the fall of 1998, while the owners of the other three properties refused permission for the removal. No properties were identified where lead levels in composite soil samples were high enough to warrant a time-critical soil removal action.

2.3 RISK-BASED SAMPLING PROGRAM

Following completion of the Phase I/Phase II sampling programs, USEPA undertook a number of additional studies in order to provide information that would help support long-term risk-based decision making at the site. One of these studies, referred to as the Risk-Based Sampling Program, collected more detailed data on metal contamination and exposure at the 18 properties that had been identified as requiring time-critical soil removal. Key elements of the program included: 1) detailed soil sampling to reveal the spatial pattern of contamination at some of the impacted properties; 2) measurement of arsenic and lead levels in several different environmental media, including indoor dust, attic dust, and garden vegetables (arsenic and lead), as well as paint and tap water (lead only); and 3) measurement of lead and arsenic levels in residents at those locations. The details of the study design are presented in USEPA (1998b), and the results are detailed in USEPA (1999a). The main findings of this program are summarized below.

2.3.1 Spatial Patterns of Contamination

One of the striking findings that emerged from the Phase I/Phase II sampling programs was that properties that were impacted by arsenic did not appear to occur in a clear spatial pattern. That is, the occurrence of high arsenic levels in soil did not appear to be associated with proximity to one or more of the smelters, and properties with elevated levels of arsenic often occurred immediately adjacent to one or more residences that were not apparently impacted.

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In order to obtain additional information on the spatial pattern of contamination both within and between yards, USEPA selected eight properties to undergo detailed soil sampling. Five of the yards were locations where Phase I/Phase II sampling indicated the arsenic concentrations were above the removal level, while three of the properties had arsenic concentrations below the removal level.

At each property, a high-density grid was established on 5-foot centers, and soil samples were collected wherever the grid node did not fall on a driveway, patio, etc. In addition, whenever access could be obtained, the sampling grid was extended 10-15 feet into adjacent properties in order to determine if there was a clear difference in contamination levels between adjacent properties. All samples were analyzed by XRF for arsenic, lead, cadmium, and zinc.

Diagrams which show the results for all four metals at all eight properties are presented in USEPA (1999a). Diagrams from this report that show the spatial patterns of arsenic and lead at two properties with high levels of arsenic contamination are shown in Figures 2-2 and 2-3. In both cases, arsenic levels vary from location to location, but are elevated across most of the yard. At property 1 (Figure 2-2), there is a fairly clear boundary between the property of concern and the adjacent properties. A similar pattern is observed at property 2 (Figure 2-3), although there are some locations where the contamination may extend somewhat into the adjacent property. The pattern of lead contaminations at these properties also showed a similar boundary effect. No clear boundary effect was observed for cadmium or zinc.

2.3.2 Contaminant Levels in Other Environmental Media

Samples of other environmental media were obtained at each removal property where access was granted. The results are summarized below.

Indoor Dust

Dust from interior living spaces were collected at 15 properties, while attic dust was collected at 9 properties. Summary statistics are presented below.

Medium	Arsenic			Lead		
	Detection Frequency	Mean (ppm)	Max (ppm)	Detection Frequency	Mean (ppm)	Max (ppm)
Interior dust (ppm)	14/15	107	172	15/15	243	1145
Attic dust (ppm)	7/9	230	499	9/9	1414	4106

Correlation analysis revealed no significant association ($p > 0.50$) between the concentration of either arsenic or lead in interior dust compared to that in outdoor soil (based on the mean of the two five-point composites that were collected in Phase II). Although this data set is too small to draw definite conclusions, the results suggest that outdoor soil is not a major determinant of arsenic or lead levels in indoor dust.

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Tap Water

Twelve properties allowed sampling and analysis of tap water for lead. Two types of water sample were collected: first flush and post-flush. Summary statistics are presented below.

Medium	Lead		
	Detect. Freq.	Mean (ug/L)	Max (ug/L)
First-flush tap water	5/12	3.2	11.4
Post flush tap water	3/12	2.5	6.0

All of these values are below the current USEPA action level for lead in drinking water (15 ug/L), and are sufficiently low that tap water is not likely to be a significant source of exposure, at least in the 12 homes sampled.

Paint

Sixteen properties authorized analysis of lead levels in paint. Concentrations were measured by XRF at multiple locations on both interior and exterior surfaces. The mean value in all interior samples was 4.2 mg/cm², with a range of 0.3 to 19.0 mg/cm². For exterior samples, the mean was 4.8 mg/cm², with a range of 0.4 to 14 mg/cm². A total of 130 out of 144 samples had values above 1 mg/cm², the national default screening level for leaded paint (HUD 1995). These data suggest that interior and/or exterior leaded paint might be a source of lead exposure in area children, either directly (by paint chip ingestion), or indirectly (by ingestion of dust or soil containing paint chips).

Garden Vegetables

Only one of the 18 properties scheduled for soil removal had a vegetable garden. At this location, one sample of potato and one sample of mint were collected. Concentrations of arsenic and lead were below the level of detection in both samples. Because so few samples were obtained, no conclusions can be drawn from this data set.

2.3.3 Biomonitoring

A total of 15 individuals residing at 6 of the properties scheduled for soil removal volunteered to have samples of hair, urine and blood analyzed for arsenic or lead. The results are summarized below. For convenience, a reference value indicating the upper end of the normal range is also presented.

Parameter	As in hair (ug/g)	As in Urine (ug/L)	Pb in Blood (ug/dL)
Detect. Frequency	1/15	0/15	14/15
Mean	0.27	8.7	2.17
Max	0.66	10	4
Reference value	1.0	20	10

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As seen, there were no cases where individuals living at the properties scheduled for soil removal had arsenic or lead levels that entered a range of concern. Although this data set is too small to draw firm conclusions, the results suggest that exposures at these locations were not of immediate health concern.

2.4 PHYSICAL-CHEMICAL CHARACTERIZATION STUDY

In addition to the Risk-Based Sampling Program described above, USEPA also undertook a study to characterize the physical and chemical attributes of the metal contamination in residential site soils, and to determine whether concentration estimates based on bulk (unsieved) soil samples were representative of concentrations in fine (sieved) samples. The design of this project is presented in USEPA (1998c), and the results are detailed in USEPA (1998d). The main findings are summarized below.

2.4.1 Concentration in Sieved and Un-Sieved Fine Soil Samples

As discussed in greater detail in Section 3, the main pathway by which humans are likely to be exposed to contaminants in soil is by incidental ingestion of soil particles adhering to the hand. Although data are limited, it is generally expected that small soil particles are more likely to adhere to the hands than coarse particles, and it is for this reason that USEPA Region VIII recommends that measurements of contaminant concentrations in soil generally be performed on samples that have been sieved to isolate the smaller particles (< 250 μ m). This sieved fraction is generally referred to as the "fine" fraction. Soil that has not been fine sieved but only coarse sieved (to remove particles larger than 10 mm) is referred to as the "bulk" sample. Studies at other sites have shown that concentrations of metals in the fine fraction can sometimes be somewhat higher (e.g, 10-30%) than in the bulk sample.

Because all of the samples collected during Phase I and Phase II were bulk samples, an investigation was performed to determine if the concentration values obtained for the bulk samples were likely to have values significantly different than if the samples had been sieved. A total of 120 samples were selected for the comparison, being sure to include samples with a wide range of arsenic and lead concentrations. All analyses were performed by XRF.

The results are shown in Figure 2-4. For lead, cadmium, and zinc, the concentration measured in the fine fraction is, on average, very nearly equal to that in the bulk fraction, with the data lying very close to the line of identity. For arsenic, there is a tendency for the concentration values in the fine fraction to be somewhat higher than in the bulk fraction, with an average difference of about 20%.

2.4.2 Speciation of Arsenic and Lead

Most metals, including arsenic and lead, can occur in a variety of different chemical and physical forms. These differences are of potential significance not only because they may help identify the source of contamination, but also because the toxicity of the metals may differ between different

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chemical forms. Therefore, USEPA undertook a study to obtain preliminary data on the chemical forms of arsenic and lead present in site soils.

The details of the sample selection, preparation and analysis are presented in USEPA (1999c). In brief, a set of 22 site soils were chosen for analysis, spanning a range of arsenic and lead concentration values. Each sample was analyzed by electron microprobe analysis (EMPA), and the number and size of different chemical forms ("phases") of arsenic and lead-bearing particles were measured. From these data, the fraction of the total mass of arsenic and lead present in each phase was calculated.

The results are shown in Figures 2-5 and 2-6. As seen, arsenic occurs mainly as arsenic trioxide, with a smaller fraction present as arsenic antimony oxide. In most samples, the majority of all arsenic-bearing particles are 5-50 um in diameter. Lead occurs in several phases, with the highest amount present as lead arsenic oxide. Other lead phases present include lead manganese oxide and lead phosphate, but these forms do not appear to increase as a function of total lead concentration, suggesting that these phases may be mainly natural in origin. In most samples, the majority of lead-bearing particles are 5-100 um in diameter.

2.5 SELECTION OF CHEMICALS OF POTENTIAL CONCERN

Chemicals of potential concern (COPCs) are chemicals which a) are present at a site, b) occur at concentrations which are or might be of health concern to exposed humans, and c) are or might be due to releases from a Superfund site. USEPA has derived a standard method for selecting COPCs at a site, as detailed in *Risk Assessment Guidance for Superfund: Human Health Evaluation Manual (Part A)* (USEPA 1989). In brief, USEPA assumes that any chemical detected at a site is a candidate for selection as a COPC, but identifies a number of methods that may be used for determining when a chemical is not of concern and may be eliminated from further consideration. Each risk assessment may choose to apply some or all of the methods identified by USEPA to select COPCs, as appropriate.

Data collected during Phase I and Phase II clearly indicated that arsenic and lead were both chemicals of potential concern at the VBI70 site. However, at that time no systematic evaluation had been performed to determine whether or not any other chemicals might also be of potential concern. For this reason, a careful review of the available data was undertaken to determine if other chemicals should be added to the list (USEPA 1999b). This review is summarized below.

2.5.1 Data Used to Select COPCs

As discussed above, most soil samples collected from the site were analyzed by XRF for only a few contaminants (mainly arsenic and lead). However, a sub-set of samples were analyzed for the full suite of 23 metals included on USEPA's Target Analyte List (TAL), and these data are the basis of the COPC selection procedure. The data consist of two sub-sets:

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- During Phase I, a total of 44 samples of soil were selected at random for TAL analysis. The chief purpose of the analysis was to assess the accuracy of the XRF measurements for arsenic and lead. Because these samples were selected *a priori* and without regard to the level of contamination, there are only 9 of these samples that contain concentrations of arsenic above 100 ppm, with the maximum value being 1,200 ppm. Thus, these samples are helpful in the COPC selection procedure, but may not necessarily represent the chemicals of concern at the most contaminated properties.
- During the Risk-Based Sampling Program, USEPA performed an intensive study of arsenic and lead levels at 8 residential properties in the study area, including 5 properties with clearly elevated arsenic levels. Two samples from each of these five properties were selected for TAL analysis, since these samples all contain high levels of arsenic (6,000 to 12,000 ppm) and are likely to reflect the contaminants most likely to be of concern.

These data are summarized in Table 2-1. In the case of copper, there is one sample whose analytical value (14,000 ppm) appears to be clearly inconsistent with all of the other 53 values (average = 37 ppm, max = 71 ppm). On this basis, the one extreme value for copper was excluded as an outlier, and screening was based on the remaining samples. All other data values were used. Non-detects were evaluated using the reported detection limit.

2.5.2 COPC Selection Process

Step 1: Eliminate Chemicals Whose Maximum Value Is Below a Level of Concern

This step involves comparing the maximum detected value in a medium to an appropriate Risk-Based Concentration (RBC). If the maximum value is less than the RBC, the chemical does not pose an unacceptable risk and can be eliminated.

The RBCs used in this evaluation were taken from USEPA's Region III Risk-Based Concentration (RBC) table for residential soil (EPA 1999c). The value of each RBC depends on the specified Target Risk level. In accord with the goal that the COPC selection process should be conservative, the Target Risk levels used in this evaluation are 1E-06 for carcinogenic chemicals and a hazard quotient (HQ) of 1.0 for noncarcinogenic chemicals.

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Table 2-2 lists the Region III RBCs for each chemical and identifies those which can and cannot be eliminated at this step. Based on this screening step, the following chemicals were eliminated:

- | | |
|-------------|-------------|
| • Aluminum | • Manganese |
| • Barium | • Mercury |
| • Beryllium | • Nickel |
| • Cadmium | • Selenium |
| • Chromium | • Silver |
| • Cobalt | • Vanadium |
| • Copper | • Zinc |

Step 2. Eliminate Beneficial Minerals

In accord with USEPA (1989), chemicals that are normal constituents of the body and the diet and are required for good health may be eliminated unless there is evidence that site-specific releases have elevated concentrations into a range where intakes would be potentially toxic. At this site, there is no reason to suspect this is the case, so the following chemicals are eliminated on this basis:

- Calcium
- Magnesium
- Potassium
- Sodium

Iron is also eliminated on this basis, since the average concentration of iron (13,400 ppm) is well below the screening level of 23,000 ppm. Additionally, only 1 of 54 samples exceeds the RBC for iron, and this only by a small amount (26,000 vs. 23,000 ppm).

Step 3. Eliminate Chemicals Whose Contribution is Minor Compared to Others

Following Steps 1 and 2, the list of chemicals remaining as potential COPCs is:

- Arsenic
- Antimony
- Lead
- Thallium

Antimony (a non-carcinogenic chemical) may be eliminated because the magnitude of the non-cancer risk which it poses is very small compared to that posed by arsenic. For example, in the 10 samples most contaminated with arsenic, the average non-cancer risk contributed by antimony is less than 1% of that contributed by arsenic. That is, if antimony were retained and the non-cancer

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risk were quantified, the risk would be less than 1% larger than if antimony were not included. Because an increment of 1% is well within the uncertainty range of the risk assessment procedure, inclusion of antimony would not change any risk interpretations and therefore is judged to be unnecessary.

Step 4. Special Investigation for Thallium

Data on thallium available from the existing TAL analyses are internally inconsistent, as shown below:

Parameter	Data Set 1	Data Set 2
Method	ICP-Trace	ICP-MS
Mean (ppm)	13.5	0.45
Max (ppm)	19	0.68
Detection Limit (ppm)	10	0.1

The basis for this internal inconsistency is not clear. One possibility is that differences in analytical methods are responsible. Data in Set 1 (collected during Phase I) utilized an analytical method (ICP-Trace, USEPA Method 6010) that had a relatively high detection limit, and most of the reported values were near that detection limit. In the second data set, thallium was analyzed by USEPA Method 6020 (ICP-MS), which has a much lower detection limit for thallium. In general, the results of the second analysis are thought to be more reliable, and are in accord with expected thallium levels in background soils. However, because it is not certain that the results from the second analysis are actually more reliable than from the first, a special study was performed in which thallium levels were measured in 10 site soils, including 6 samples from Set 1 (previously analyzed by ICP-trace) and 4 samples from Set 2 (previously analyzed by ICP-MS). Each of the samples were analyzed for thallium by three analytical methods:

- Inductively Coupled Plasma Atomic Emission Spectroscopy [ICP-trace]
(EPA SW-846 Method 6010B)
- Inductively Coupled Plasma-Mass Spectrometry [ICP-MS]
(EPA SW-846 Method 6020)
- Graphite Furnace Atomic Absorption Spectroscopy [GFAA]
(EPA SW-846 Method 7841)

The results of this analysis are provided in Table 2-3. A comparison of thallium levels in site soils as reported in past and present studies clearly indicate that results contained in the Phase I Investigation report (UOS 1998a) are biased high and are not reliable, with all of the 10 present site soil measurements having thallium values below 1 ppm. Based on the Region III (EPA 1999c) risk-based concentration for thallium in soil (5 ppm), it is concluded that thallium is not in a range of potential concern, and therefore it was eliminated as a COPC.

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2.5.3 Summary: Chemicals Selected as COPCs at VBI70

Based on the methods and data detailed above, the COPCs selected for quantitative evaluation at the VBI70 site are arsenic and lead. All other chemicals are either not of concern or are present at levels which contribute minimal risk compared to arsenic.

2.6 PHASE III INVESTIGATION

Results from the Phase I/Phase II sampling programs, supplemented with the data and findings from the Risk-Based Sampling Program and the Physical Chemical Characterization Program, indicated that there are properties present in the VBI70 site where arsenic and/or lead could be in a range of health concern to exposed humans. However, because of the absence of any clear spatial pattern of soil contamination, the identity and location of such properties can not be reliably predicted using traditional approaches. For this reason, USEPA undertook a large-scale sampling program designed to obtain data that would help evaluate health risks to residents in the area. This program is referred to as the Phase III investigation. The investigation consisted of four main parts:

- Sampling of residential yard soils
- Sampling of indoor dust at residences
- Sampling of residential vegetable gardens (vegetables and soil)
- Supplemental sampling of soil at local schools and parks

The details of the Phase III sampling program are presented in USEPA (1999d).

Phase III was implemented in two parts. The first part, referred to as Phase IIIa, focused mainly on properties (including residences, schools, and parks) which had not been investigated in Phases I or II. The second part, referred to as Phase IIIb, consisted of re-sampling at properties that had previously been sampled in Phase I or II, but for which the data were judged to be too limited to support clear risk-management decision making.

At the time of the preparation of this public review draft, the results from the Phase IIIa effort are available, but the results from the Phase IIIb effort are not yet complete. The Phase IIIa results are summarized below.

2.6.1 Residential Soil Sampling

A total of 30 surface soil (0-2 inch) grab samples were collected from each property where access was granted. These 30 samples were combined into three composite samples, each containing 10 grab samples. The composites were prepared by combining every third grab sample, such that each composite represents an independent estimate of the yard-wide mean concentration. All composite samples were dried and thoroughly mixed, and then analyzed for arsenic and lead by XRF.

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The total number of properties targeted in Phase IIIa was about 2,600. Of these, a total of 1,637 granted USEPA authority to collect samples, and samples were successfully collected at a total of 1,548 properties. Summary statistics, based on average values at each property and stratified by neighborhood, are summarized in Table 2-4. The distributions of arsenic and lead concentrations across the entire site are shown graphically in Figure 2-7.

For arsenic, most properties (1,270 out of 1,548) have average concentrations of 50 ppm or less, with 126 properties between 50-100 ppm, 106 between 100-200 ppm, and 46 above 200 ppm.

For lead, 1389 properties have mean lead concentrations lower than 400 ppm, with 153 between 400-800 ppm and 6 higher than 800 ppm.

The relationship between the concentration of lead and arsenic in residential yard soil samples is shown in Figure 2-8. As seen, there is a statistically significant correlation ($p < 0.001$) between lead and arsenic, but this high correlation is largely the result of the large number of samples ($N = 5721$), and the correlation explains only a small amount of the variability ($R^2 = 0.095$). Inspection of the figure indicates that samples with high lead values (e.g., those above 500 ppm) occur over a wide range of arsenic values, and are not associated predominantly with those where arsenic is also clearly elevated (e.g., above 100 ppm). This indicates that the main sources of lead and the main sources of arsenic in yard soil are not likely to be the same.

2.6.2 Residential Dust Sampling

As discussed in greater detail in Section 3, one pathway by which residents may be exposed to contaminants in soil is by transport of outdoor soil into the house where it combines with other sources to form house dust. When data are absent, USEPA often assumes that the concentration of contaminants in house dust is the same as in yard soil. However, studies at other sites have shown that dust levels of metals are often lower in indoor dust than in outdoor soil. Therefore, USEPA undertook a study to define the relationship between arsenic and lead levels in soil and dust at this site. The details of the sampling and analysis plan are presented in the Phase III Project Plan (USEPA 1999d). In brief, a total of 76 properties were selected for study. These properties were chosen to provide for a range of arsenic and lead levels in soil, and to provide for spatial representativeness across the site. Samples were collected in October and November, 1999.

The results are shown in Figure 2-9. In the case of lead, two dust samples were excluded as outliers because they contained lead at concentration values (2,000 ppm and 9,900 ppm) that were much higher than that observed in yard soil (268 ppm and 320 ppm, respectively). The source of the high dust lead at these two locations is not known, but could be associated with leaded paint.

As seen, there is only a weak correlation between the level of either arsenic or lead in paired soil and dust samples ($R^2 = 0.14$ to 0.18 , respectively). Nevertheless, the slopes of both regression

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lines are statistically different from zero ($p < 0.01$), with best estimate parameter values as follows:

$$\text{Arsenic: } C_{\text{dust}} = 0.06 \cdot C_{\text{soil}} + 11$$

$$\text{Lead: } C_{\text{dust}} = 0.34 \cdot C_{\text{soil}} + 150$$

2.6.3 Residential Garden Sampling

Another pathway by which residents might be exposed to soil-related contaminants is ingestion of vegetables grown in home gardens that contain contaminated soil. In order to obtain site-specific data on this potential exposure route, USEPA collected 72 samples of different types of garden vegetables from 19 different properties around the site. The detailed data are presented in Appendix A. The mean concentrations of arsenic and lead were 0.044 and 0.15 ug/g wet weight, respectively. There was no apparent difference in concentration as a function of vegetable type (exposed, protected, root).

At each location where a vegetable sample was collected, a co-located sample of garden soil was also collected. The results for these garden soil samples are also presented in Appendix A. For arsenic, the concentrations in garden soils were generally lower than in the corresponding yard soils, with no significant relationship between the two (Figure 2-10, upper panel). For lead, garden soils also tended to be lower than for yard soils, but there was a weak correlation between the two (Figure 2-10, lower panel). These data suggest that garden soils are not equivalent to yard soils, presumably because most gardens are amended by addition of soil, peat, fertilizer, etc. The weak correlation for lead suggests that the base soil in the garden may tend to come from the yard, but the absence of a correlation for arsenic suggests that whatever the source is for yard soil does not apply to garden soil.

The relationships between the concentration of arsenic and lead in garden vegetables and in the co-located garden soil are shown graphically in Figure 2-11. As seen, there is very little tendency for the concentration of either lead or arsenic in garden vegetables to increase as a function of the concentration of in garden soil, although there is a slight (and statistically significant) trend for arsenic (slope = 0.002 mg/kg ww per mg/kg in soil). The slope for lead is very close to zero and is not statistically significant.

2.6.4 Sampling at Schools and Parks

As noted above, data on the levels of arsenic and lead in surface soil were collected at a number of schools and parks during the Phase I investigation. However, in most cases only a few samples were collected from each location, and not all schools and parks were sampled. Therefore, the Phase III Sampling and Analysis Plan included collection of 15-30 supplemental surface soil grab samples from each school and park within the site (the number depending on the size of the

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property). In Phase 3a, samples were collected from 10 schools and one park. The results are shown in Table 2-5. The remainder of all schools and parks will be samples in Phase 3b.

As seen, for the locations sampled during Phase 3a, concentrations of arsenic are generally low, with average values ranging from 11-15 ppm, and maximum values less than 30 ppm. An exception to this pattern occurred at one property owned by a school (location code S12). At this location, two values significantly higher than expected were detected (1517 ppm and 70 ppm)¹. These values occur adjacent to each other, and are surrounded by values of 17-23 ppm. This suggests there might be a small "hot spot" at this location. Because no children are presently exposed at this location, this is not a source of immediate concern. However, USEPA plans to investigate this location further and take action as appropriate.

2.7 DATA SELECTED FOR USE IN THIS RISK ASSESSMENT

The data from the Phase III sampling program were selected for use in this risk assessment because 1) all Phase III data were collected in accordance with project plans that were developed with careful consideration of the Data Quality Objectives (DQOs) needed to support risk assessment calculations, and 2) all data collected during Phase III are accompanied by thorough Quality Assurance (QA) data that allow detailed evaluation of the reliability of the data.

A detailed review of these quality assurance data (USEPA 2000a) reveal that the data collected are of high quality, with adequate accuracy and precision to support a reliable evaluation of human health risk.

Data collected during Phase I/Phase II were not used because they were collected only with the intent of identifying locations that exceeded the removal action levels, and were not intended to support risk calculations or remedial decision making. More specifically, data from Phase I/Phase II were not used because 1) many samples had elevated detection limits for arsenic, 2) the sampling density at each property was too low, and/or 3) sampling locations were not clear. However, despite these limitations, it is clear that the data from Phase I/Phase II and from Phase III are generally similar, each indicating the occurrence of scattered properties with elevated levels of lead and/or arsenic. The USEPA is currently using the Phase III sampling and analysis plan to collect additional samples from properties originally sampled during Phase I/Phase II so that reliable decisions regarding the need for remediation at these properties can be made.

¹ These two samples were re-analyzed in triplicate to confirm the data. The mean values for the re-analyzed samples were 978 ppm and 114 ppm, respectively.

TABLE 2-1 DATA USED TO SELECT COPCs

Analyte	N	Detection Frequency	Summary Statistics		
			Min	Max	Mean
ALUMINUM	54	100%	4900	15000	8761
ANTIMONY	54	22%	2.2	54	6.8
ARSENIC	54	93%	5	9940	543
BARIUM	54	100%	91	1000	251
BERYLLIUM	54	98%	0.3	1.1	0.7
CADMIUM	54	100%	0.9	19	5.9
CHROMIUM	54	100%	7.2	99	22
COBALT	54	98%	1.0	7.0	4.6
COPPER (a)	53	100%	12	71	37
LEAD	54	100%	36	3550	712
MANGANESE	54	100%	160	560	323
MERCURY	54	93%	0.1	11	1.0
NICKEL	54	100%	5.9	96	11
SELENIUM	54	19%	0.3	10	9
SILVER	54	69%	0.3	3	0.7
THALLIUM	54	89%	0.2	19	11
VANADIUM	54	100%	13	42	21
ZINC	54	100%	84	3680	499
CALCIUM	54	100%	1900	41000	6757
IRON	54	100%	7900	26000	13405
MAGNESIUM	54	100%	1400	4100	2400
POTASSIUM	54	100%	1400	4100	2350
SODIUM	54	5%	300	440	304

(a) Excludes one value (14,000 ppm) that is considered anomalous

TABLE 2-2 COMPARISON OF MAXIMUM VALUES IN SOIL
TO SOIL SCREENING LEVELS (a)

Analyte	Maximum Conc (ppm)	Region III Soil Screening Level (ppm)	Potential COPC?
ALUMINUM	15000	78400	no
ANTIMONY	54	31	yes
ARSENIC	9940	0.43	yes
BARIUM	1000	5500	no
BERYLLIUM	1.1	160	no
CADMIUM	19	78	no
CALCIUM	41000	--	no
CHROMIUM	99	230	no
COBALT	7.0	4700	no
COPPER (a)	71	3100	no
IRON	26000	23000	yes
LEAD	3550	400	yes
MAGNESIUM	4100	--	no
MANGANESE	560	1600	no
MERCURY	11	23	no
NICKEL	96	1600	no
POTASSIUM	4100	--	no
SELENIUM	10	390	no
SILVER	3	390	no
SODIUM	440	--	no
THALLIUM	19	5.5	yes
VANADIUM	42	550	no
ZINC	3680	23000	no

(a) Soil screening levels based on USEPA (1999a)

TABLE 2-3 COMPARISON OF PAST AND PRESENT DATA FOR THALLIUM IN SOIL

Sample ID	Thallium Concentration (mg/kg)				
	Past Results		Present Study		
	ICP-MS	ICP-T	ICP-T	ICP-MS	GFAA
C4690CYB-064	0.63		10 U	0.70	0.50 U
C4690CYB-046E	0.20		10 U	0.10	0.50 U
C4711THF-001	0.33		10 U	0.30	0.50 U
C4771VIN-001	0.33		10 U	0.30	0.50 U
D4145FIB10		12	10 U	0.20 U	0.50 U
D4715GYF10		17	10 U	0.30	0.50 U
D4050FIB10		11	10 U	0.20	0.50 U
D4701JOS10		10 U	10 U	0.10 U	0.50 U
D4780CBB10		16	10 U	0.50	0.80
D4785CLF10		15	10 U	0.20	0.50 U

U - Target analyte not detected

TABLE 2-4 SUMMARY STATISTICS FOR PHASE IIIa SOIL SAMPLES

ARSENIC

Statistic	Clayton	Cole	Five Points	Globeville	Swansea/Elyria	All
N	644	601	14	22	267	1548
5th	5.5	5.5	5.5	5.5	5.5	5.5
25th	5.5	7.7	10.3	8.3	5.5	5.5
50th	10.3	11.8	16.4	10.8	9.0	10.9
75th	40.5	23.9	21.9	18.3	23.9	30.7
90th	109.7	91.4	28.4	33.6	93.1	98.4
95th	175.9	150.7	39.7	44.8	140.3	161.9
Max	609.6	659.8	59.2	70.9	430.6	659.8

LEAD

Statistic	Clayton	Cole	Five Points	Globeville	Swansea/Elyria	All
N	644	601	14	22	267	1548
5th	79	135	170	170	82	87
25th	111	216	383	226	119	135
50th	147	284	436	282	177	204
75th	201	367	549	331	281	309
90th	286	452	687	383	409	402
95th	348	515	764	424	479	475
Max	1131	1004	835	782	922	1131

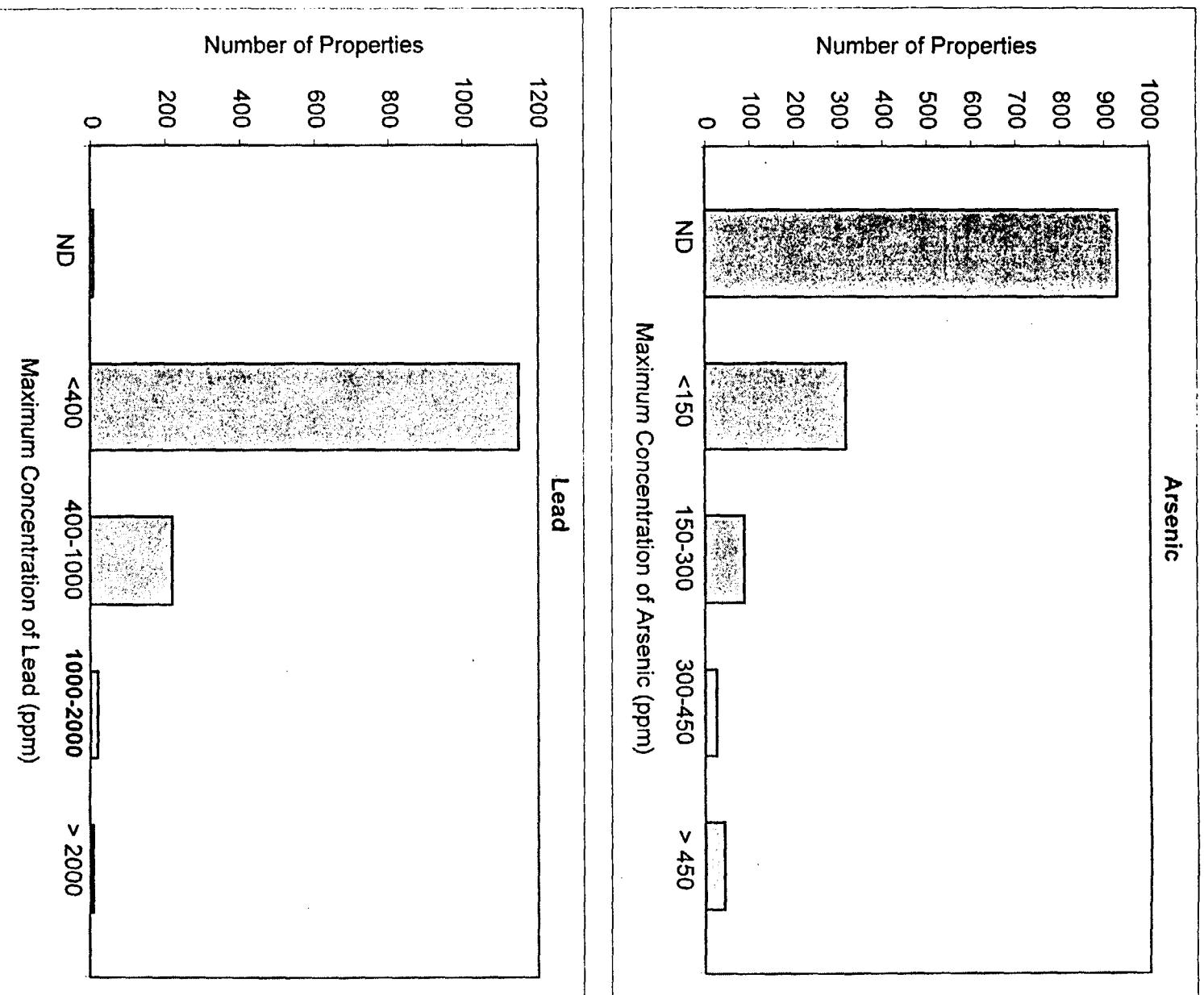
All values are based on the mean value observed at each property

TABLE 2-5 PHASE IIIa SOIL DATA FOR SCHOOLS AND PARKS

Category	Property Code	N	Arsenic (ppm)			Lead (ppm)		
			Mean*	Max	Min	Mean	Max	Min
School	S1	30	11	12	11	95	164	52
	S2	30	12	19	11	200	628	55
	S3	30	11	11	11	67	126	52
	S4	15	11	13	11	83	102	57
	S5	30	11	11	11	72	255	52
	S6	15	11	11	11	69	95	52
	S7	30	11	12	11	104	245	52
	S8	30	67	1517	11	310	1811	88
	S9	30	11	18	11	223	567	61
	S10	30	12	19	11	235	359	127
Park	P1	30	14	21	11	215	398	52

*Mean arsenic concentration is not adjusted

FIGURE 2-1 PHASE I/PHASE II SOIL GRAB SAMPLE DATA



Notes:

ND = Not detected

FIGURE 2-2 SPATIAL DISTRIBUTION OF CONTAMINANTS -- PROPERTY 1

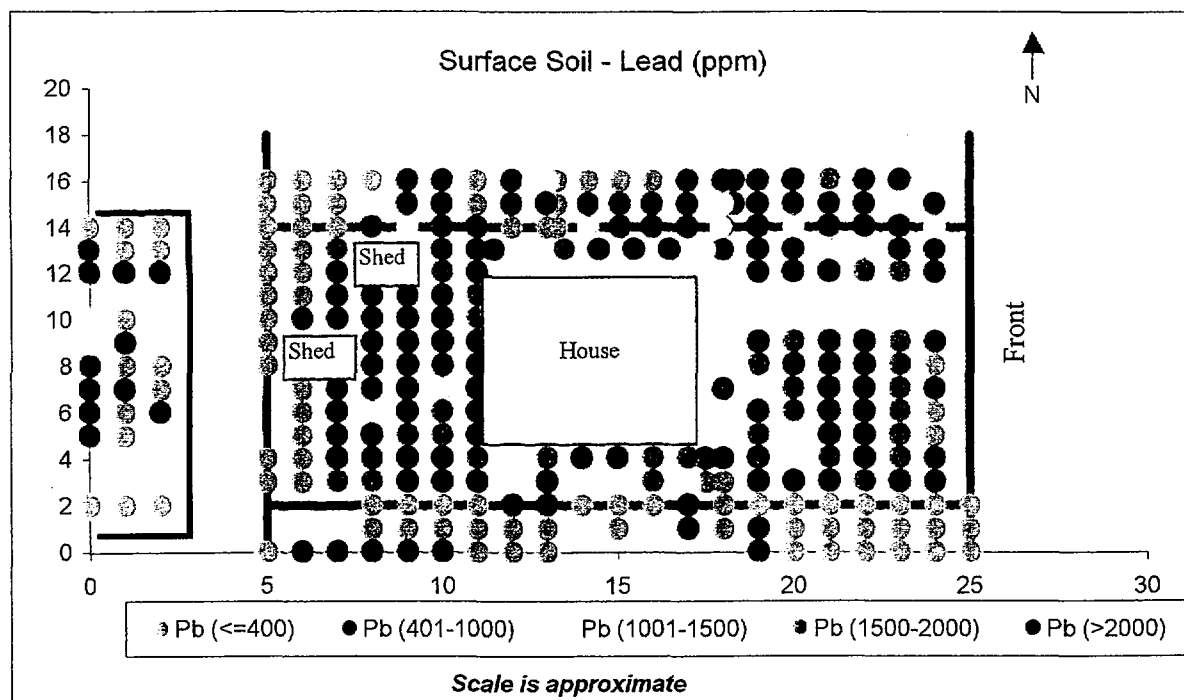
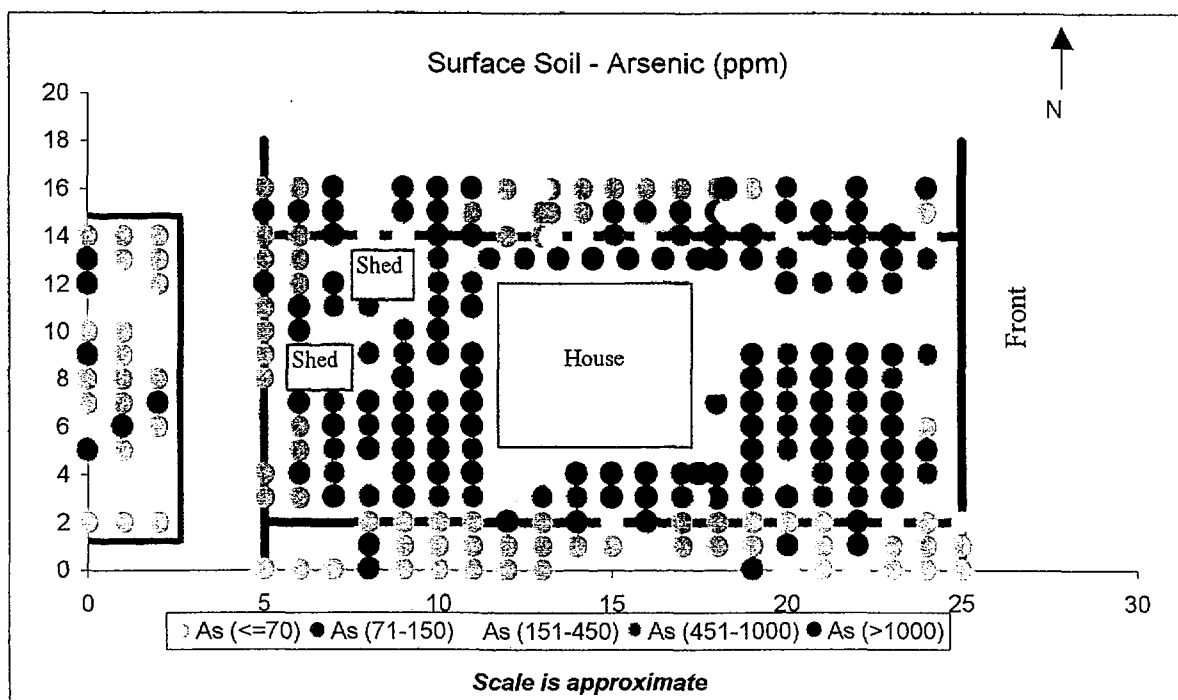


FIGURE 2-3 SPATIAL DISTRIBUTION OF CONTAMINANTS -- PROPERTY 2

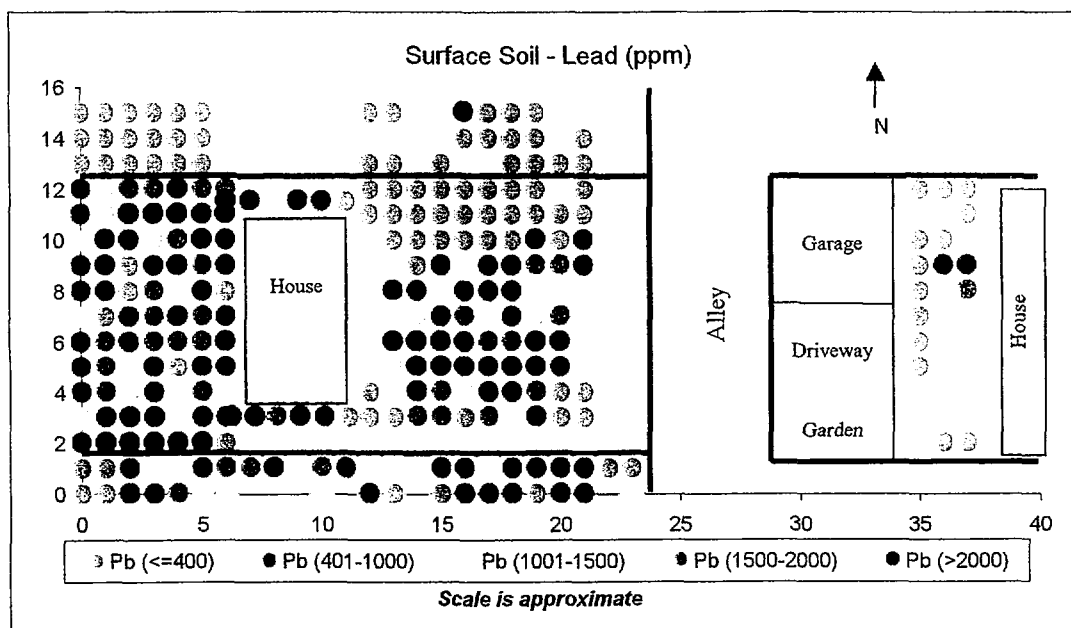
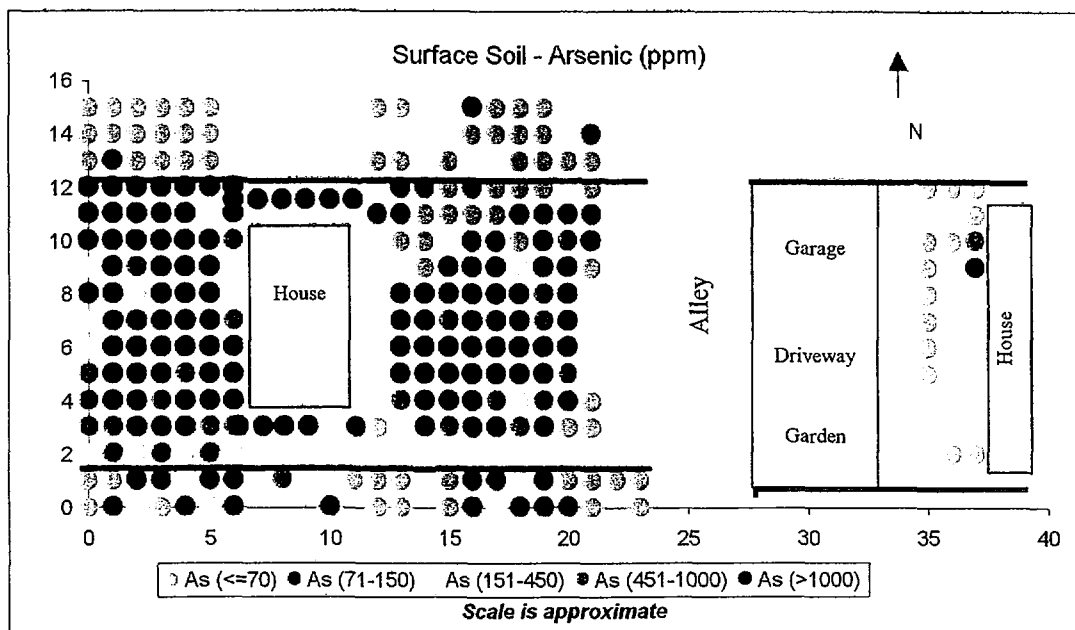


FIGURE 2-4 COMPARISON OF CONCENTRATIONS IN BULK AND FINE SOIL

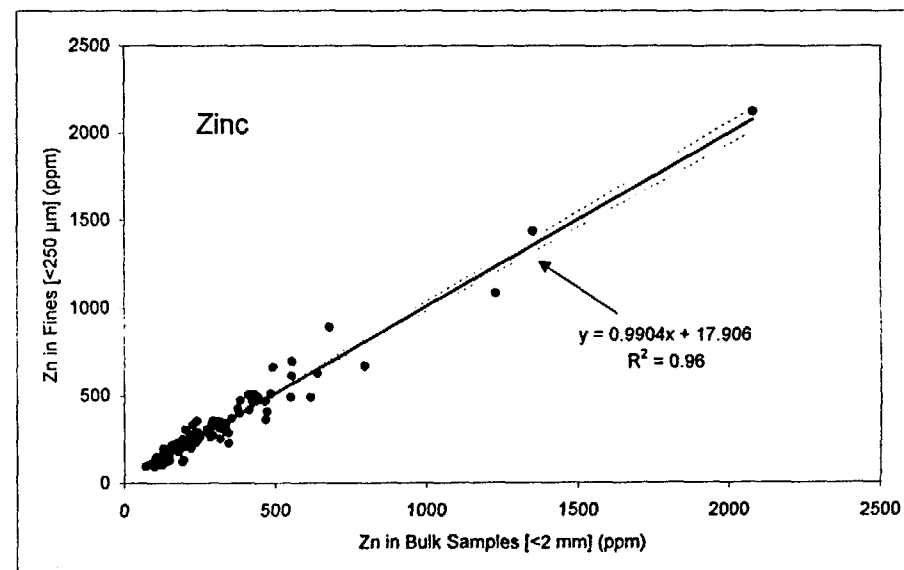
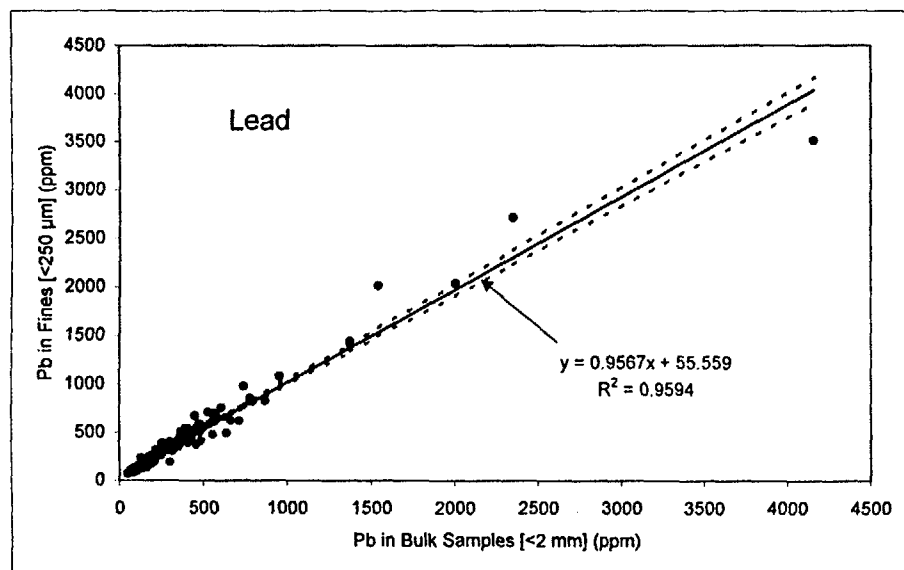
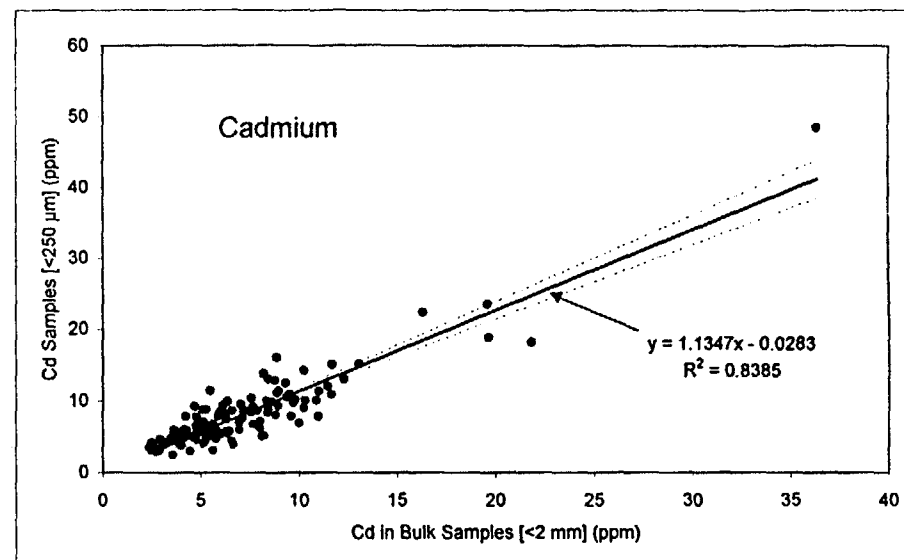
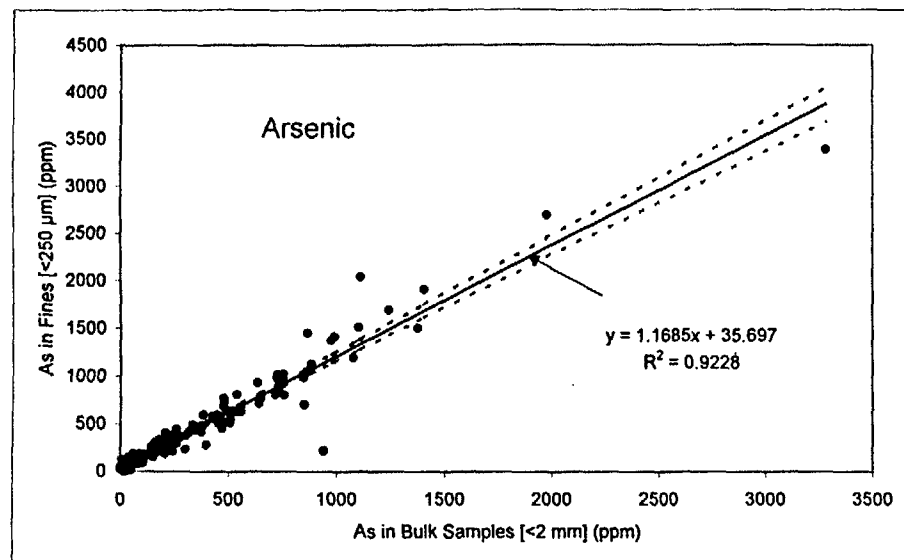


FIGURE 2-5 CHEMICAL FORMS OF ARSENIC IN SITE SOILS

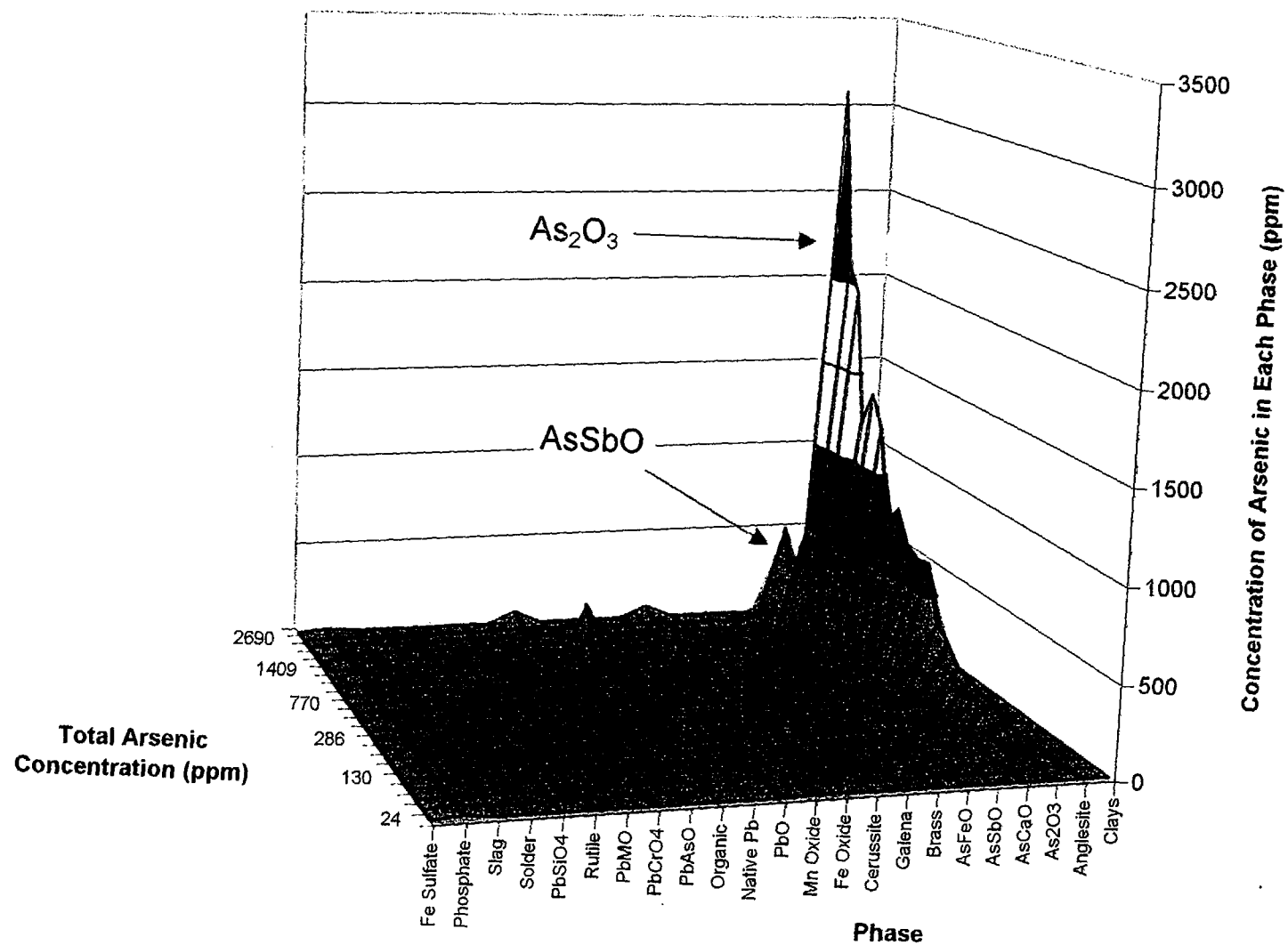


FIGURE 2-6 CHEMICAL FORMS OF LEAD IN SITE SOILS

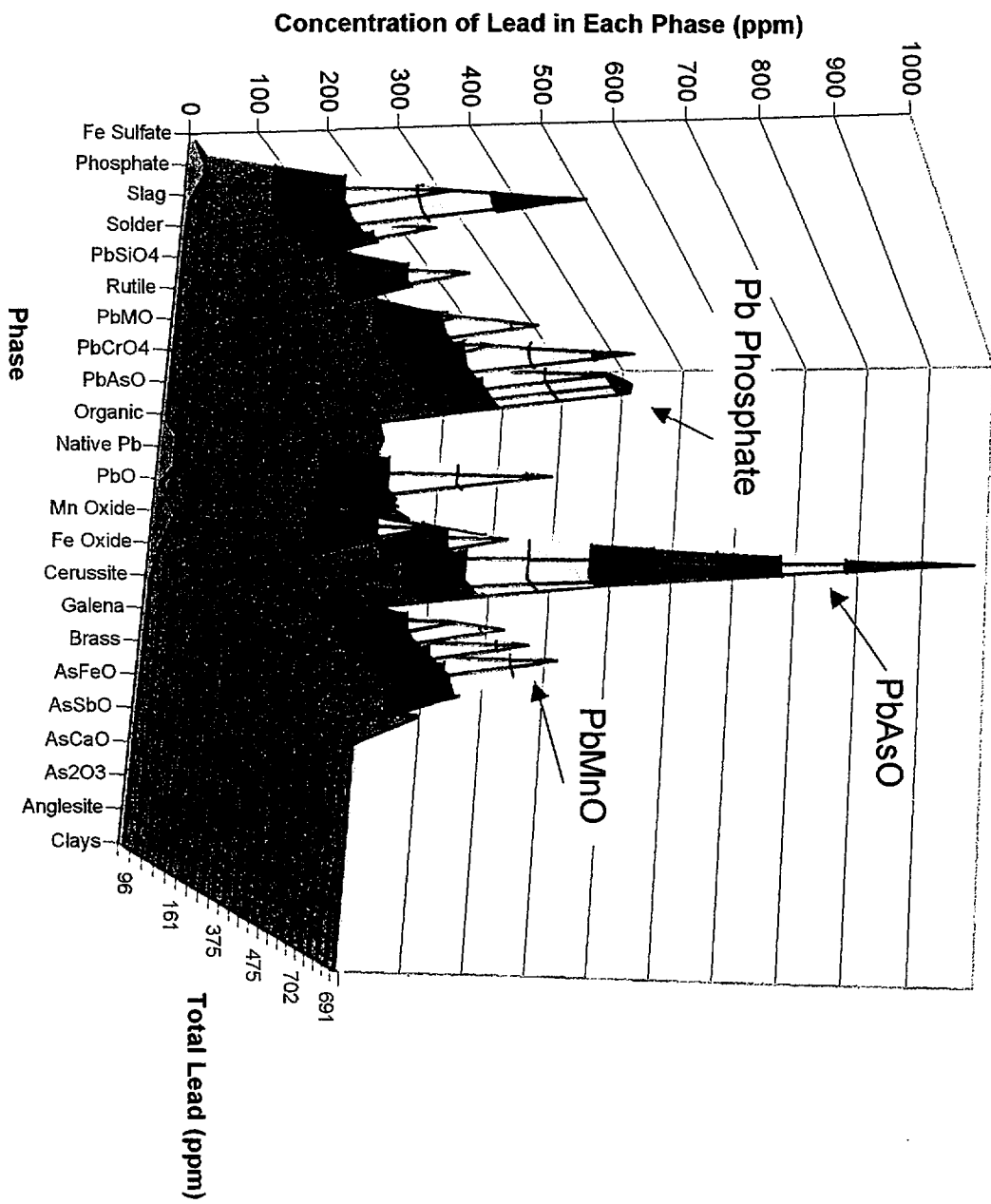


FIGURE 2-7 DISTRIBUTION OF PROPERTY MEAN CONCENTRATIONS IN PHASE IIIa SOIL SAMPLES

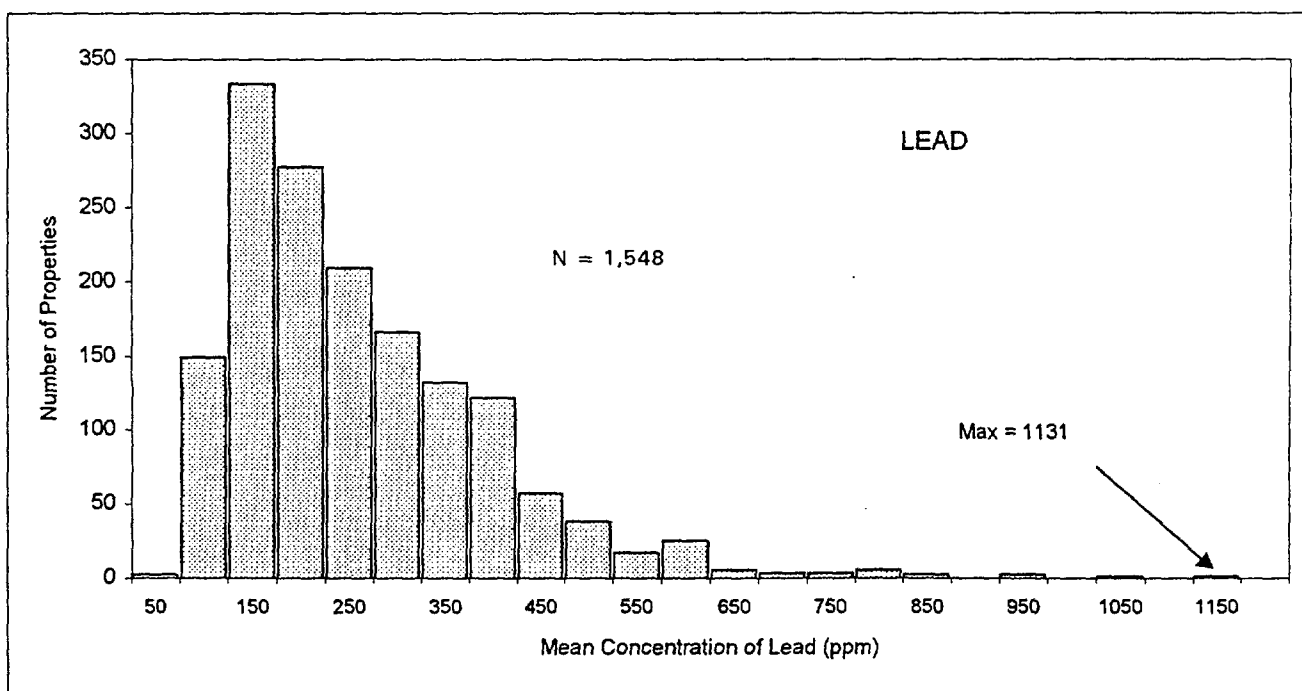
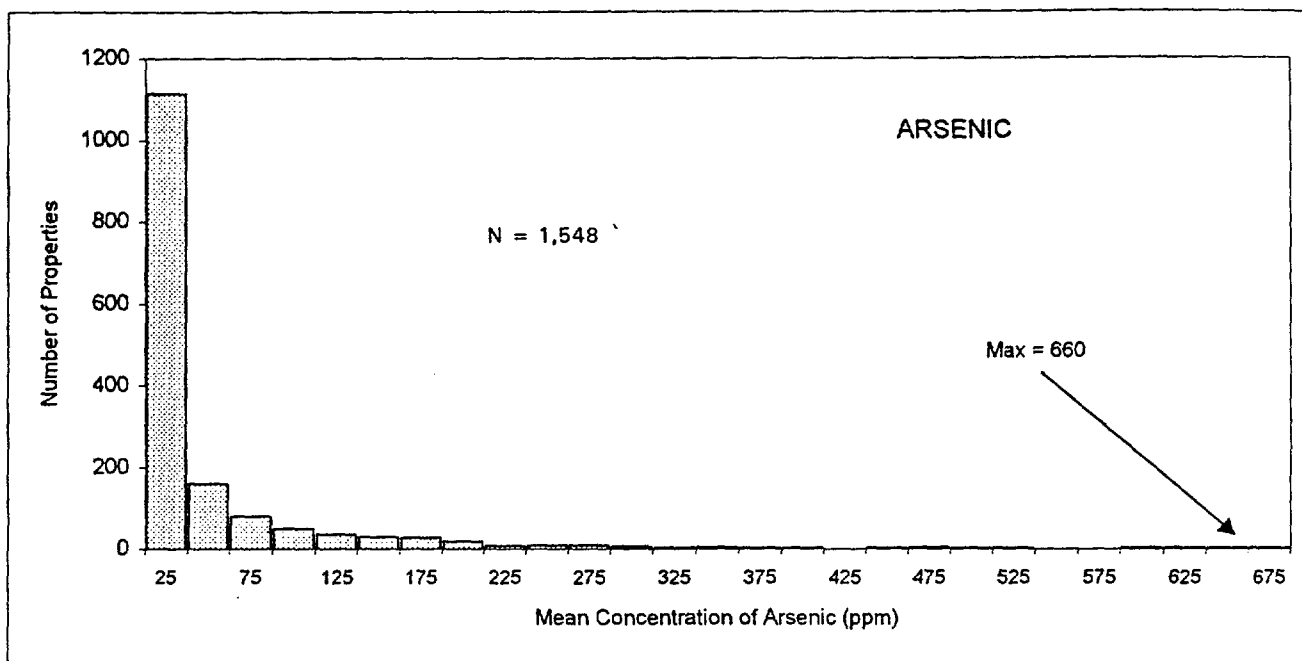


FIGURE 2-8 CORRELATION BETWEEN LEAD AND ARSENIC IN PHASE IIIa SOILS

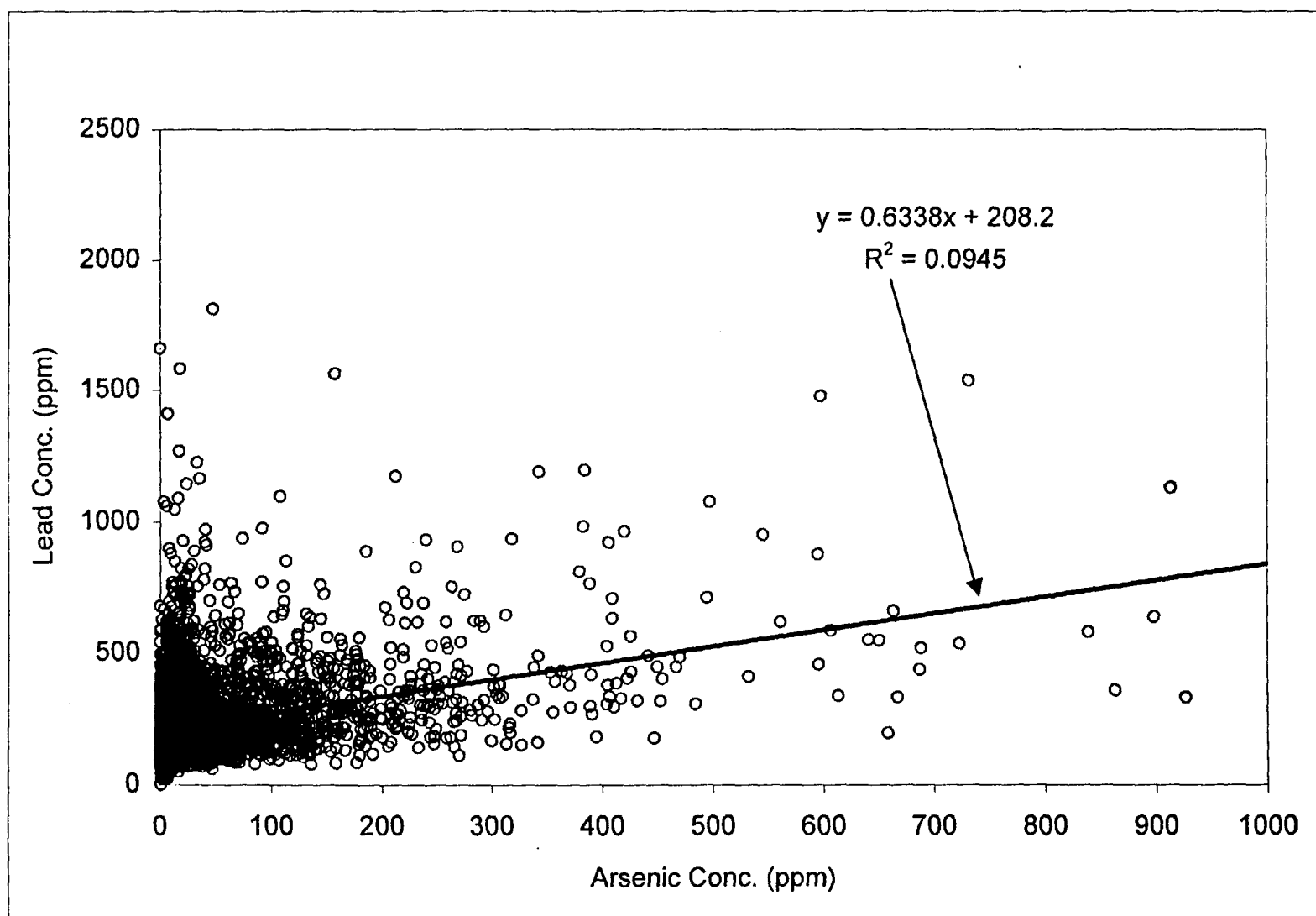


FIGURE 2-9 RELATION BETWEEN CONCENTRATIONS IN INDOOR DUST AND OUTDOOR SOIL

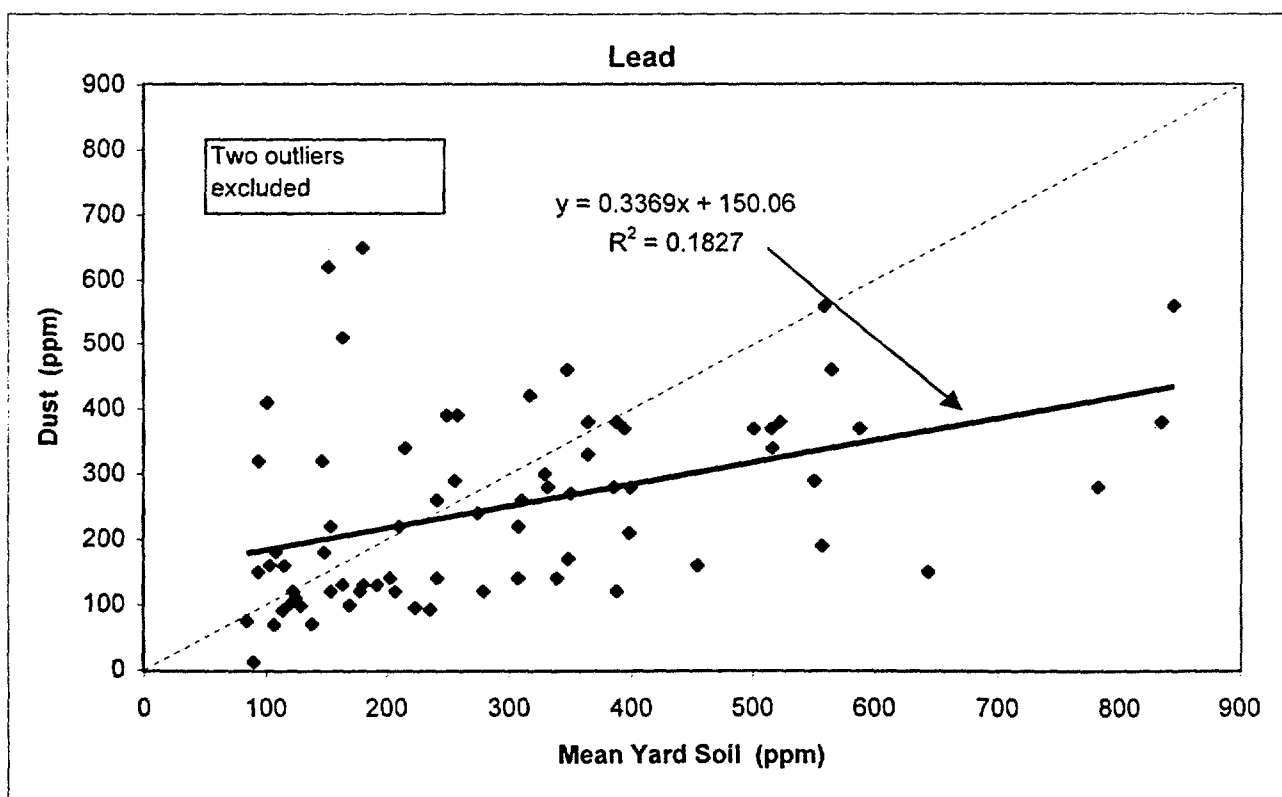
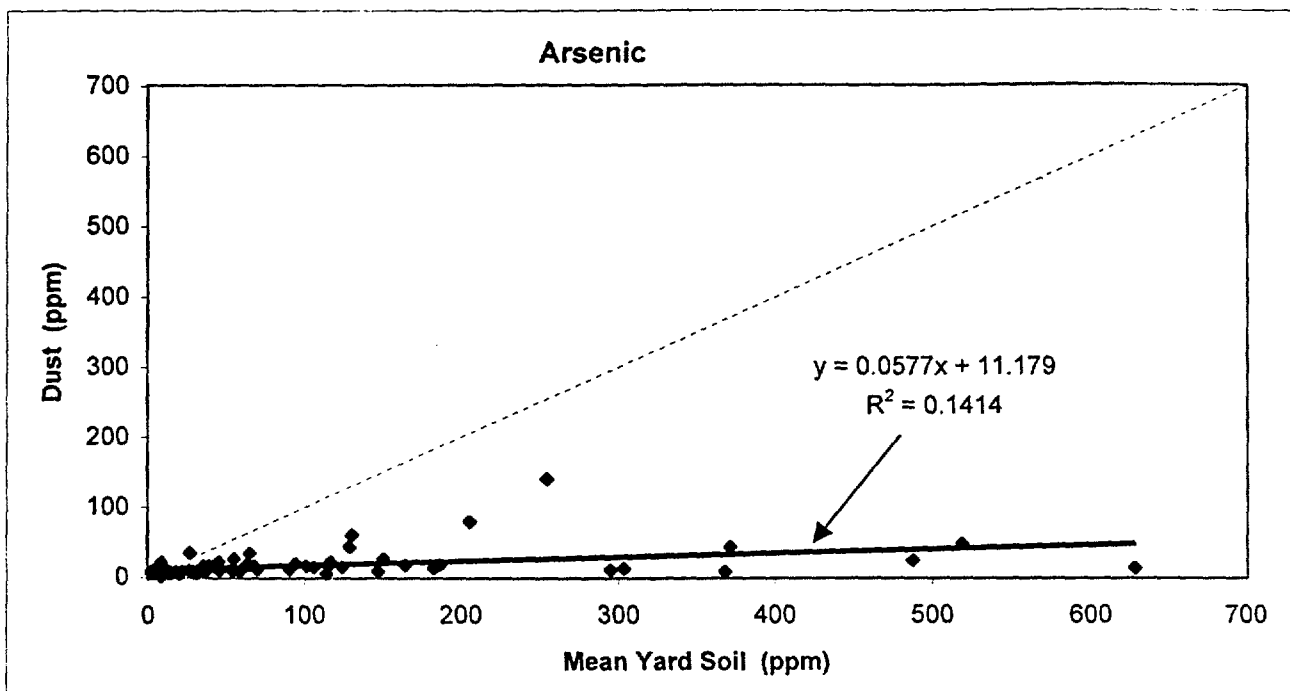
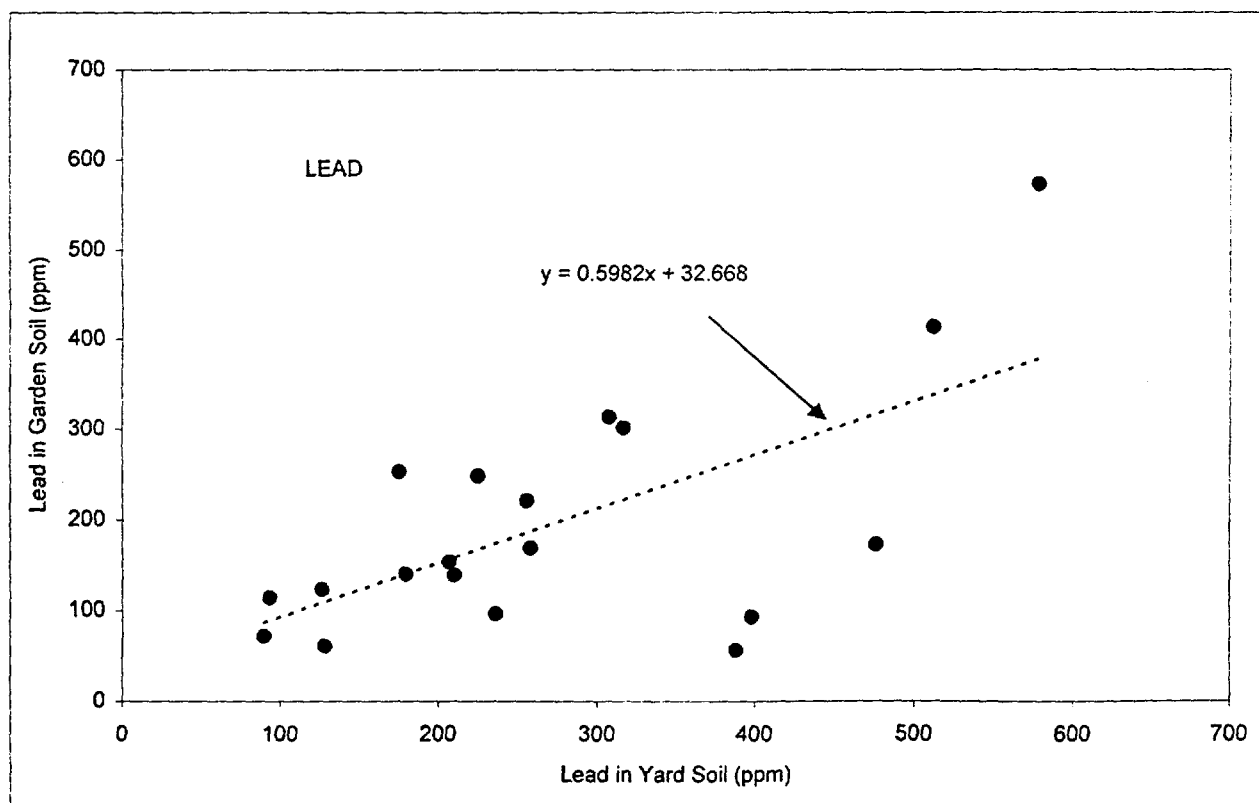
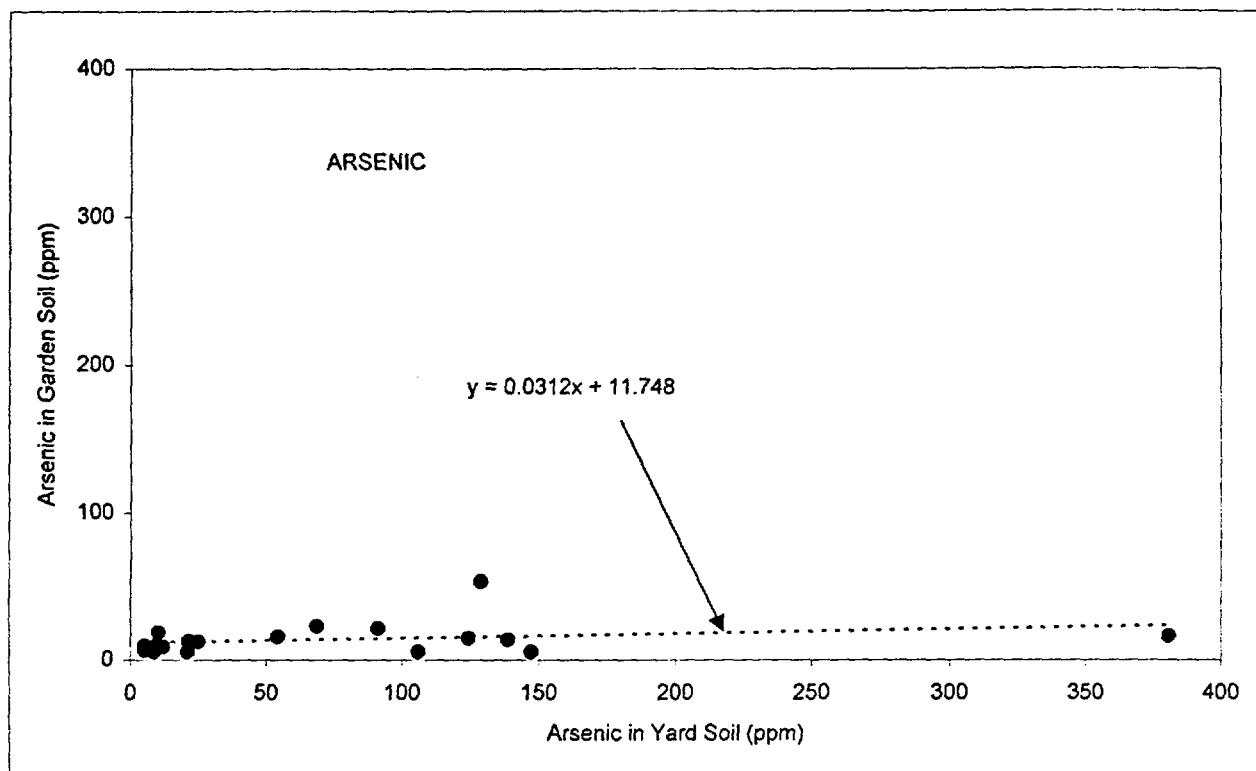
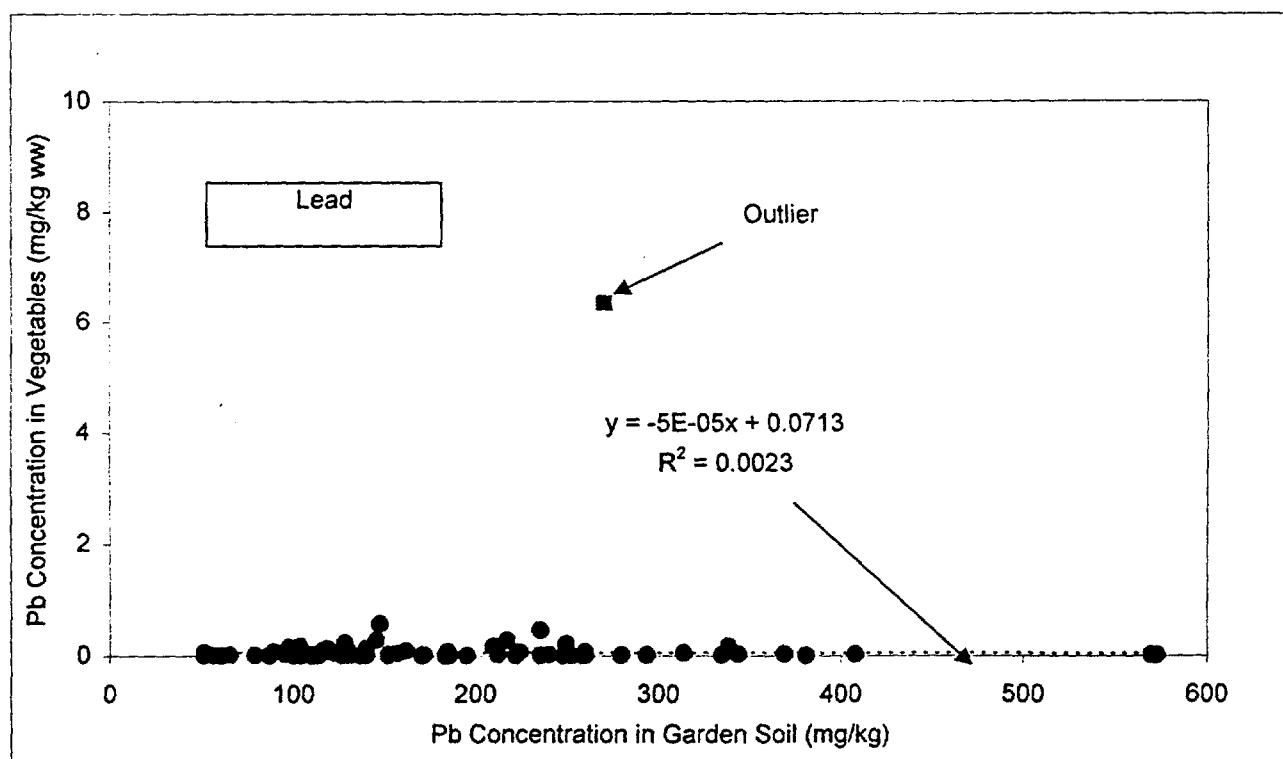
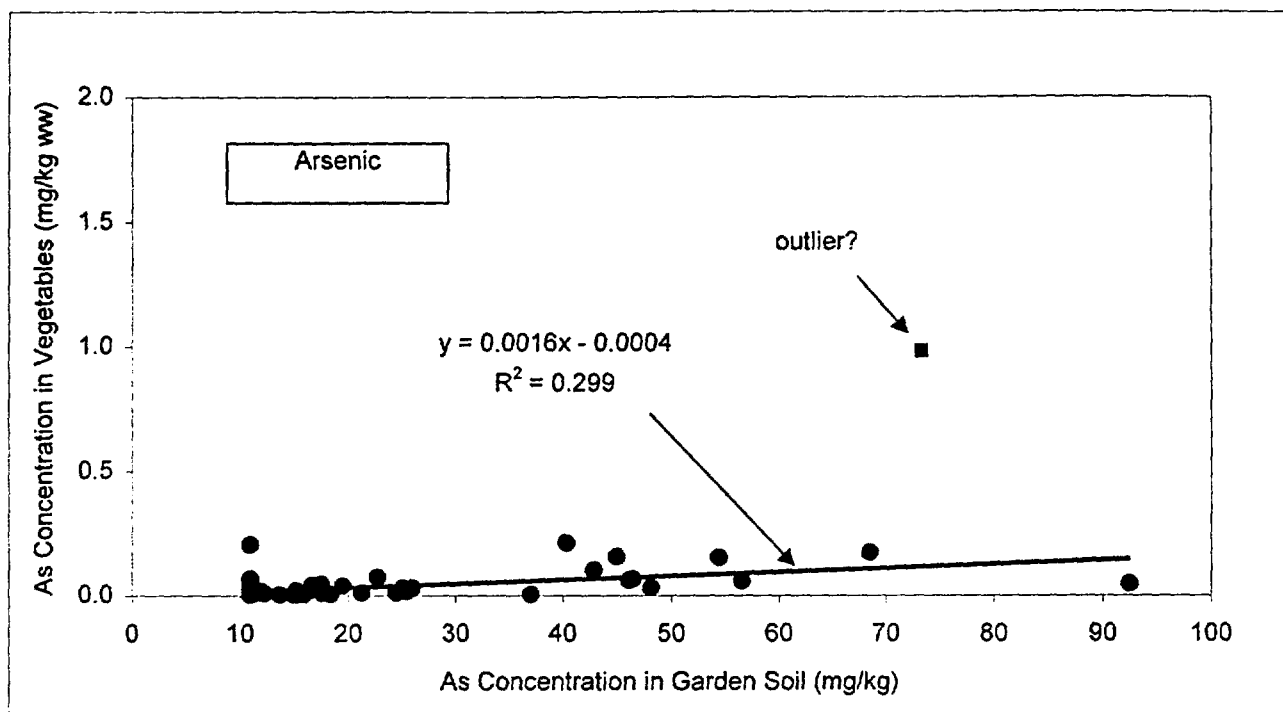


FIGURE 2-10 RELATION BETWEEN CONTAMINANTS IN GARDEN SOIL AND YARD SOIL



Mean Yard and Garden Soil

FIGURE 2-11 RELATION BETWEEN CONCENTRATIONS IN GARDEN
VEGETABLES AND GARDEN SOIL



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SECTION 3 EXPOSURE ASSESSMENT

Exposure is the process by which humans come into contact with chemicals in the environment. In general, humans can be exposed to chemicals in a variety of environmental media (e.g., soil, dust, water, air, food), and these exposures can occur through one or more of several pathways (ingestion, dermal contact, inhalation). Section 3.1 provides a discussion of possible pathways by which area residents and workers might come into contact with contaminants present in outdoor soil. Section 3.2 describes the basic methods used to estimate the amount of chemical exposure which humans may receive from direct and indirect contact with contaminants derived from outdoor soil.

3.1 CONCEPTUAL SITE MODEL

Figure 3-1 presents a conceptual model showing the main pathways by which contaminants present in surface soil may come into contact with area residents. Exposure scenarios that are considered most likely to be of concern are shown in Figure 3-1 by boxes containing a solid circle, and greatest attention is focused on these pathways. Pathways which are judged to contribute only occasional and minor exposures are shown by boxes with an open circle. Incomplete pathways (i.e., those which are not thought to occur) are shown by open boxes. The following sections present a more detailed description of each of these exposure scenarios, and presents the basis for concluding that some pathways are minor.

3.1.1 Potential Sources

The source of soil contamination at residential properties at the VBI70 site is not yet established. Two alternative hypotheses (which are not mutually exclusive) are that the contamination observed in yard soils is due to a) smelter-related releases (either airborne fallout from historic operations and/or bulk transport of contaminated waste material), or b) application of some sort of pesticide or lawn care product (e.g., PAX is a crabgrass killer that contains both arsenic and lead). Studies are currently underway to obtain data that may help distinguish between these alternatives (USEPA 1999e).

3.1.2 Migration Pathways

Regardless of the source, the current medium of chief concern is soil. Metals in soil tend to have relatively low mobility (they are not volatile, and usually do not tend to migrate extensively in soil). Rather, contaminants in soil tend to move mainly by bulk transport, either by a) wind-blown transport of suspended soil particles in air, b) surface water run-off of contaminated soil in ditches, etc., or c) hauling of bulk material from one location to another.

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3.1.3 Exposed Populations and Potential Exposure Scenarios

There are a number of different groups or populations of humans who may come into contact with smelter-related contaminants in area soils. This includes area residents and workers, as well as individuals who may be exposed at area schools or parks. The following text describes the scenarios which are considered plausible for each population, and identifies which are likely to be most important and which are sufficiently minor that they need not be evaluated quantitatively.

3.2 PATHWAY SCREENING

3.2.1 Residential Exposures

Incidental Ingestion of Soil

Few people intentionally ingest soil. However, it is believed that most people (especially children) do ingest small amounts of soil that adhere to the hands or other objects placed in the mouth. In addition, outdoor soil can enter the home and mix with indoor dust, which may also be ingested during meals or during hand-to-mouth activities. This exposure pathway is often one of the most important routes of human intake, so it was selected for quantitative evaluation.

Dermal Contact with Soil

Residents can get contaminated soil on their skin while working or playing in their yard. Even though information is limited on the rate and extent of dermal absorption of metals in soil across the skin, most scientists consider that this pathway is likely to be minor in comparison to the amount of exposure that occurs by soil and dust ingestion. This view is based on the following concepts: 1) most people do not have extensive and frequent direct contact with soil, 2) most metals tend to bind to soils, reducing the likelihood that they would dissociate from the soil and cross the skin, and 3) ionic species such as metals have a relatively low tendency to cross the skin even when contact does occur. These presumptions are supported by screening level calculations which indicate that dermal exposure of most metals is likely to be no larger (and probably much lower) than absorption due to soil ingestion (see Appendix B). Based on these considerations, along with a lack of data to allow reliable estimation of dermal uptake of metals from soil, Region VIII generally recommends that dermal exposure to metals in soils not be evaluated quantitatively (EPA 1995a). Therefore, this pathway was not evaluated quantitatively in this risk assessment.

Inhalation of Soil/Dust in Air

Particles of contaminated soil or dust become resuspended in air, and residents may breathe those particles both inside and outside their house. However, screening level calculations (presented in Appendix B) based on conservative estimates of soil release to air indicate that for residents, inhalation of particles is likely to be a small source of risk (less than 0.2%) compared to incidental ingestion of soil. Based on this, it was concluded that inhalation exposure is a sufficiently minor

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contributor to exposure and that it need not be included in the quantitative evaluation of residential exposure.

Ingestion of Home-Grown Vegetables

If a resident raises vegetables or fruits in a home garden that contains contaminated soil, some contamination may be taken up from the soil into the vegetable. If so, the resident would be exposed when those vegetables were consumed. Although data on exposure by this pathway are limited, some studies suggest that the pathway may contribute a significant fraction of the total exposure. Therefore, this pathway was selected for quantitative evaluation.

3.2.2 Workplace Exposures

Workers at commercial or industrial locations within the site boundary may be exposed to soil while working in outdoor locations, so incidental ingestion, inhalation of particulates and/or dermal contact may occur. As is the case with residents, ingestion exposure is the most important of these exposure routes. Although no soil samples have been collected from commercial properties at the VBI70 site, extensive sampling has been performed at commercial properties in the vicinity of the Globe plant (EnviroGroup 2000). This sampling has revealed that, with the exception of some properties close to the plant that have been impacted by slag or by direct surface water runoff, there is very little evidence of significant contamination at commercial properties:

Parameter	Arsenic	Lead
Number of commercial properties sampled	345	345
Average concentration (ppm)	20	145
Highest average concentration (ppm)	96	1064
Risk-based concentration for workers (see Appendix C)	382	1545

Because there is no known reason why commercial properties in the vicinity of the Globe site should be less contaminated than commercial properties in the vicinity of the VBI70 site, these data are assumed to be representative of what would be obtained if sampling were to proceed at commercial properties within the VBI70 site. However, screening-level calculations presented in Appendix C show that these concentrations are below a level of concern to workers. On this basis, it is concluded that sampling at commercial properties and detailed quantitative risk calculations for workers are not needed at the VBI70 site. Therefore, the worker population is not evaluated further in this risk assessment.

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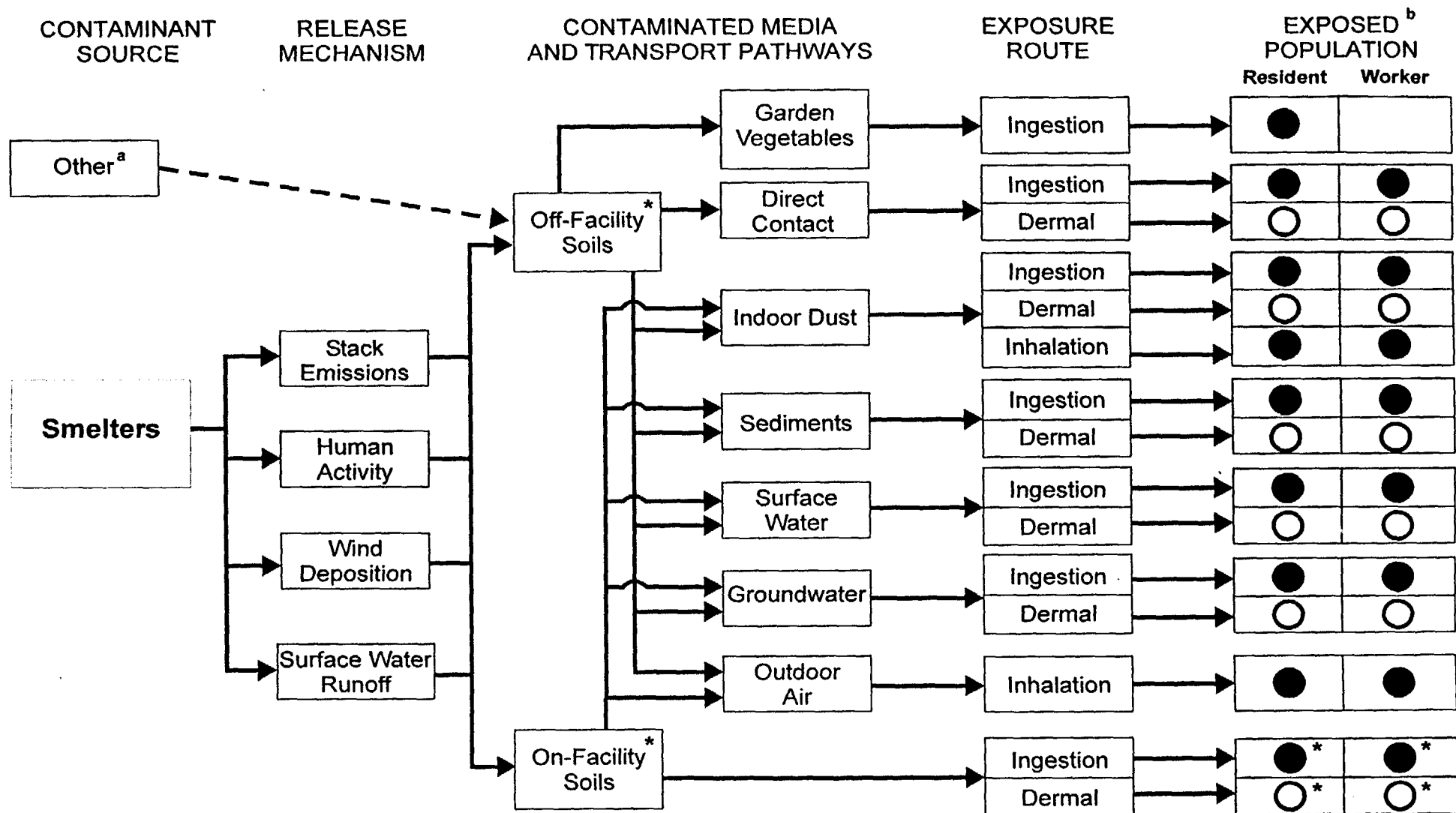
3.3 SUMMARY OF PATHWAYS OF PRINCIPAL CONCERN




Based on the evaluations above, the following pathways are judged to be of sufficient potential concern to warrant quantitative exposure and risk analysis:

Population	Medium and Exposure Route
Resident	Incidental ingestion of soil and dust in and about the home and yard Ingestion of home-grown vegetables

Other exposure pathways are judged to be sufficiently minor that further quantitative evaluation is not warranted.

FIGURE 3-1 CONCEPTUAL SITE MODEL
POTENTIAL HUMAN EXPOSURE PATHWAYS AT VASQUEZ BLVD./I-70 SITE



-  = Pathway is not complete
-  = Pathway is complete, but minor; qualitative evaluation
-  = Pathway is complete and could be significant; quantitative evaluation

* "On-Facility" exposure is only at the former Omaha-Grant and Argo sites.

^a - Other sources may be historical smelters, other active smelters & arsenical pesticides.

^b - The work group will refine the list of exposed populations as the risk assessment proceeds and

SECTION 4

QUANTIFICATION OF EXPOSURE AND RISK FROM ARSENIC

4.1 OVERVIEW

The USEPA has established standard methods for estimating the level of exposure and risk to residents from a variety of chemical contaminants in soil. These methods are employed below to estimate the exposure and risk to residents at the VBI70 site from arsenic in soil. Whenever possible, site specific data are used in preference to non-site specific default assumptions.

Because the approach used to evaluate exposure and risk from lead is somewhat different than that used for arsenic, the assessment of lead risks is presented separately in Section 5.

4.2 QUANTIFICATION OF EXPOSURE

4.2.1 Basic Equation

The amount of a chemical which is ingested, inhaled, or taken up across the skin is referred to as "intake" or "dose", and is usually calculated using an equation of the following general form:

$$DI = C \cdot (IR/BW) \cdot (EF \cdot ED/AT)$$

where:

DI =	Daily intake of chemical (mg of chemical per kg of body weight per day)
C =	Concentration of the chemical in the contaminated environmental medium (soil, dust, etc.) to which the person is exposed. The units are mg of chemical per unit of environmental medium (e.g., mg/kg for soil, food, etc.).
IR =	Intake rate of the contaminated environmental medium. The units are usually kg/day for solid media (soil, dust, food).
BW =	Body weight of the exposed person (kg).
EF =	Exposure frequency (days/year). This describes how often a person is likely to be exposed to the contaminated medium over the course of a typical year.

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ED = Exposure duration (years). This describes how long a person is likely to be exposed to the contaminated medium during their lifetime.

AT = Averaging time (days). This term specifies the length of time over which the average dose will be calculated. Usually, two different averaging times are considered:

- "Chronic" exposure includes averaging times on the scale of years (typically ranging from 7 years to 70 years). This exposure duration is used when assessing the non-cancer risks from chemicals of potential concern.
- "Lifetime" exposure employs an averaging time of 70 years. This exposure interval is selected when evaluating cancer risks.

In some cases (when the concentration of contaminants is sufficiently high that short-term exposures might be of concern), a separate evaluation of "subchronic" exposure (typically from 1 to 7 years) or "sub-acute" exposure (typically from several weeks to several months) may also be performed.

Note that the last three factors (EF, ED, AT) combine to yield a factor between zero and one. Values near 1.0 indicate that exposure is nearly continuous over the specified averaging period, while values near zero indicate that exposure occurs only rarely.

For mathematical convenience, the general equation for calculating dose is often written as:

$$DI = C \cdot HIF$$

where:

HIF = Human Intake Factor. This term describes the average amount of an environmental medium contacted by the exposed person each day. The value of HIF is typically given by:

$$HIF = (IR/BW) \cdot (EF \cdot ED/AT)$$

The units of HIF are kg/kg-day for solid media such as soil, dust, and food.

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4.2.2 Variability and Uncertainty in Exposure Calculations

For every exposure pathway of potential concern, it is expected that there will be differences between different individuals in the concentration of chemical to which they are exposed, as well as differences in intake rates, body weights, exposure frequencies and exposure durations. Thus, there is normally a wide range of average daily intakes between different members of an exposed population. Because of this, all daily intake calculations must specify what part of the range of doses is being estimated. Typically, attention is focused on two different parts of the exposure distribution:

Average or "Central Tendency" Exposure (CTE) is either the arithmetic mean or the median exposure. It is calculated using the average values for all of the exposure parameters.

Reasonable Maximum Exposure (RME) is the highest exposure that is reasonably expected to occur at a site. The intent of the RME is to estimate a conservative exposure case that is still within the range of possible exposures. This is done by using a combination of upper-bound estimates for some exposure parameters and average estimates for some exposure parameters.

This variability in exposure between different members of the population should not be confused with the difficulties that are often encountered in attempting to estimate either CTE or RME daily chemical intake levels. These difficulties arise because there are usually insufficient data to accurately define key exposure parameters such as typical and upper bound intake rates, exposure frequencies and exposure durations. Thus, the choice of values for average and upper-bound intakes are often rather uncertain. In addition, there is usually uncertainty regarding the true mean concentration of a chemical in a medium at an exposure area. Because of this uncertainty, the USEPA typically recommends that, for chemicals such as arsenic, the exposure point concentration (EPC) that is used to calculate exposure and risk be based on either the 95% upper confidence limit (UCL) of the mean concentration or the maxim concentration (whichever is lower) (USEPA 1989). Note that this approach is used for both the CTE and the RME exposure scenarios (USEPA 1992a).

4.2.3 Source of Exposure Parameters

The USEPA has collected a wide variety of data and has performed a number of studies to help establish reasonable values for many human exposure parameters. The chief sources of these standard default values are the following documents:

1. Risk Assessment Guidance for Superfund (RAGS). Volume I. Human Health Evaluation Manual (Part A). USEPA 1989.

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2. Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors". USEPA 1991a.
3. Superfund's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure. Draft. USEPA 1993a.
4. Exposure Factors Handbook. Volumes I to III. USEPA 1997.

However, for some parameters, there is no guidance and there are few or no data to support the selection of CTE or RME values, so professional judgement and input from community members were utilized in some cases.

4.2.4 Quantification of Exposure of Residents

Soil and Dust Ingestion

Based on the assumption that the concentration of contaminants is approximately equal in outdoor yard soil and indoor house dust, the USEPA usually evaluates residential exposure to soil and dust in a single step. The basic equation is as follows:

$$DI_{sd} = C_{sd} \left(\frac{IR_{sd}}{BW} \cdot \frac{EF_{sd} \cdot ED}{AT} \right)$$

Both chronic and lifetime average intake rates are time-weighted to account for the possibility that an exposed individual may begin exposure as a child (EPA 1989a, 1991a, 1993a), as follows:

$$TWA - DI_{sd} = C_{sd} \left(\frac{IR_c}{BW_c} \cdot \frac{EF_c \cdot ED_c}{(AT_c + AT_a)} + \frac{IR_a}{BW_a} \cdot \frac{EF_a \cdot ED_a}{(AT_c + AT_a)} \right)$$

where:

TWA-DI_{sd} = Time-weighted Daily Intake from ingestion of soil and dust (mg/kg-d)

C_{sd} = Concentration of chemical in soil and dust (mg/kg)

IR = Intake rate (kg/day) when a child (IR_c) or an adult (IR_a)

BW = Body weight (kg) when a child (BW_c) or an adult (BW_a)

EF = Exposure frequency (days/yr) when a child (EF_c) or an adult (EF_a)

ED = Exposure duration (years) when a child (ED_c) or an adult (ED_a)

AT = Averaging time (days) while a child (AT_c) or an adult (AT_a)

Default values and assumptions recommended by USEPA (1989, 1991a, 1993a, 1997) for evaluation of chronic and lifetime residential exposure to soil and dust are listed below:

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Exposure Parameter	CTE		RME	
	Child	Adult	Child	Adult
IR (kg/day)	1E-04	5E-05	2E-04	1E-04
BW (kg)	15	70	15	70
EF (days/yr)	234	234	350	350
ED (years)	2	7	6	24
AT (noncancer effects) (days)	2·365	7·365	6·365	24·365
AT (cancer effects) (days)	--	70·365	--	70·365

Based on the exposure parameters above, the time-weighted HIFs for chronic and lifetime exposure of residents to soil and dust are as follows:

Residential Exposure to Soil plus Dust	HIF _{sd} (kg/kg-d)	
	CTE	RME
TWA-chronic (non-cancer)	1.3E-06	3.7E-06
TWA-lifetime (cancer)	1.7E-07	1.6E-06

However, as noted in Section 2, studies at a number of sites have revealed that the concentration of metals such as lead and arsenic is often not as high in indoor dust as in outdoor soil. In this situation, it is necessary and appropriate to evaluate exposure to soil and dust separately, as follows:

$$DI_{sd} = C_s \cdot HIF_s + C_d \cdot HIF_d$$

where:

C = Concentration in soil (C_s) or in dust (C_d)

HIF = Human Intake Factor for soil (HIF_s) or dust (HIF_d)

In general, the concentration of contaminants in dust can be expressed as a function of the concentration in outdoor soil using the following equation:

$$C_d = D0 + ksd \cdot C_s$$

where:

D0 = Concentration in dust (ppm) that is not attributable to yard soil

ksd = Fraction of indoor dust that is derived from outdoor soil

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If f_s is defined as the fraction of total intake that is soil, the equation above can be re-written as:

$$DI_{sd} = C_s \cdot f_s \cdot HIF_{sd} + (D0 + ksd \cdot C_s) \cdot (1 - f_s) \cdot HIF_{sd}$$

Data are sparse on the relative amounts of soil and dust ingestion by residents, but limited data support the view that total intake is composed of about 45% soil and 55% dust in children (EPA 1994a). By extrapolation, this ratio is also assumed to apply to resident adults. Thus:

$$f_s = 0.45$$

As discussed in Section 2.5.2, in order to derive a reliable site-specific estimate of the relation between yard soil and indoor dust, paired samples of yard soil and indoor dust were collected at 74 properties at the site. These data are presented in Figure 2-8. For arsenic, the best estimate of the relation between soil and dust is given by the equation:

$$C_d = 0.06 \cdot C_s + 11$$

That is, $D0 = 11$ ppm and $ksd = 0.06$.

Vegetable Ingestion

The basic equation for evaluation of exposure from ingestion of home-grown vegetables or native vegetation is as follows:

$$DI_{gv} = C_{gv} \cdot IR_{gv} \cdot \left(\frac{EF_{gv} \cdot ED}{AT} \right)$$

where:

DI_{gv} = Average daily intake of chemical from home-grown garden vegetables (mg/kg-day)

C_{gv} = Concentration in garden vegetables (mg/kg wet weight)

IR_{gv} = Average intake rate of home-grown garden vegetables (kg wet weight per kg body weight per day)

EF_{gv} = Exposure frequency to home-grown garden vegetables (days/yr)

ED = Exposure duration (years)

AT = Averaging time (days)

A number of studies on the intake of homegrown garden vegetables are summarized in the Exposure Factors Handbook (EPA 1997). Intake rates vary as a function of several parameters, including geographic region and age. For this evaluation, intake rates were based on seasonally-adjusted lifetime mean values of home-grown garden vegetable intake by people living in the western region of the United States. Time-weighted averaging of intakes across childhood and adulthood was not used since the lifetime average value is essentially identical to the calculated time-weighted value. These exposure parameters are summarized below:

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Parameter	Average	RME
IR (kg wet weight/kg body wt/day)	4.92E-04	5.04E-03
EF (days/yr)	350	350
ED (years)	9	30
AT (noncancer effects) (days)	9·365	30·365
AT (cancer effects) (days)	70·365	70·365

Based on these exposure parameters, the HIF values for exposure of residents to home-grown vegetables are as follows:

Residential Exposure to Home-Grown Garden Vegetables	HIF _{gv} (kg ww/kg-d)	
	Average	RME
Chronic (non-cancer)	4.7E-04	4.8E-03
Lifetime (cancer)	6.1E-05	2.1E-03

4.3 TOXICITY ASSESSMENT

4.3.1 Overview

The basic objective of a toxicity assessment is to identify what adverse health effects a chemical causes, and how the appearance of these adverse effects depends on dose. In addition, the toxic effects of a chemical frequently depend on the route of exposure (oral, inhalation, dermal) and the duration of exposure (subchronic, chronic or lifetime). Thus, a full description of the toxic effects of a chemical includes a listing of what adverse health effects the chemical may cause, and how the occurrence of these effects depends upon dose, route, and duration of exposure.

The toxicity assessment process is usually divided into two parts: the first characterizes and quantifies the non-cancer effects of the chemical, while the second addresses the cancer effects of the chemical. This two-part approach is employed because there are typically major differences in the time-course of action and the shape of the dose-response curve for cancer and non-cancer effects.

Non-Cancer Effects

Essentially all chemicals can cause adverse health effects if given at a high enough dose. However, when the dose is sufficiently low, typically no adverse effect is observed. Thus, in characterizing the non-cancer effects of a chemical, the key parameter is the threshold dose at

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which an adverse effect first becomes evident. Doses below the threshold are considered to be safe, while doses above the threshold are likely to cause an effect.

The threshold dose is typically estimated from toxicological data (derived from studies of humans and/or animals) by finding the highest dose that does not produce an observable adverse effect, and the lowest dose which does produce an effect. These are referred to as the "No-observed-adverse-effect-level" (NOAEL) and the "Lowest-observed-adverse-effect-level" (LOAEL), respectively. The threshold is presumed to lie in the interval between the NOAEL and the LOAEL. However, in order to be conservative (protective), non-cancer risk evaluations are not based directly on the threshold exposure level, but on a value referred to as the Reference Dose (RfD). The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

The RfD is derived from the NOAEL (or the LOAEL if a reliable NOAEL is not available) by dividing by an "uncertainty factor". If the data are from studies in humans, and if the observations are considered to be very reliable, the uncertainty factor may be as small as 1.0. However, the uncertainty factor is normally at least 10, and can be much higher if the data are limited. The effect of dividing the NOAEL or the LOAEL by an uncertainty factor is to ensure that the RfD is not higher than the threshold level for adverse effects. Thus, there is always a "margin of safety" built into an RfD, and doses equal to or less than the RfD are nearly certain to be without any risk of adverse effect. Doses higher than the RfD may carry some risk, but because of the margin of safety, a dose above the RfD does not mean that an effect will necessarily occur.

Cancer Effects

For cancer effects, the toxicity assessment process has two components. The first is a qualitative evaluation of the weight of evidence that the chemical does or does not cause cancer in humans. Typically, this evaluation is performed by the USEPA, using the system summarized in the table below:

Category	Meaning	Description
A	Known human carcinogen	Sufficient evidence of cancer in humans.
B1	Probable human carcinogen	Suggestive evidence of cancer incidence in humans.
B2	Probable human carcinogen	Sufficient evidence of cancer in animals, but lack of data or insufficient data from humans.
C	Possible human carcinogen	Suggestive evidence of carcinogenicity in animals.
D	Cannot be evaluated	No evidence or inadequate evidence of cancer in animals or humans.

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For chemicals which are classified in Group A, B1, B2, or C, the second part of the toxicity assessment is to describe the carcinogenic potency of the chemical. This is done by quantifying how the number of cancers observed in exposed animals or humans increases as the dose increases. Typically, it is assumed that the dose response curve for cancer has no threshold, arising from the origin and increasing linearly until high doses are reached. Thus, the most convenient descriptor of cancer potency is the slope of the dose-response curve at low dose (where the slope is still linear). This is referred to as the Slope Factor (SF), which has dimensions of risk of cancer per unit dose.

Estimating the cancer Slope Factor is often complicated by the fact that observable increases in cancer incidence usually occur only at relatively high doses, frequently in the part of the dose-response curve that is no longer linear. Thus, it is necessary to use mathematical models to extrapolate from the observed high dose data to the desired (but unmeasurable) slope at low dose. In order to account for the uncertainty in this extrapolation process, USEPA typically chooses to employ the upper 95th confidence limit of the slope as the Slope Factor. That is, there is a 95% probability that the true cancer potency is lower than the value chosen for the Slope Factor. This approach ensures that there is a margin of safety in cancer as well as noncancer risk estimates.

4.3.2 Toxicity Summary for Arsenic

The toxic effects of arsenic have been reasonably well established, based mainly on studies of humans exposed to elevated levels of arsenic from a variety of sources. The findings from these studies are summarized briefly below.

Noncarcinogenic Effects

Oral exposure to high doses of arsenic produces marked irritation of the gastrointestinal tract, leading to nausea and vomiting. Symptoms resulting from chronic ingestion of lower doses of arsenic often begin with a vague weakness and nausea. As exposure continues, symptoms become more characteristic and may include signs such as diarrhea, vomiting, anemia, injury to blood vessels, damage to kidney and liver, and impaired nerve function that leads to "pins and needles" sensations in the hands and feet. The most diagnostic sign of chronic arsenic exposure is an unusual pattern of skin abnormalities, including dark and white spots and a pattern of small "corns," especially on the palms and soles (ATSDR 1998).

The average daily intake of arsenic that produces these effects varies from person to person. In a large epidemiological study in Taiwan, Tseng et al. (1968) reported skin and vascular lesions in humans exposed to ingested arsenic doses of 0.014 mg/kg-day or higher. Intake was through the drinking water. These effects were not observed in a control population ingesting 0.0008 mg/kg-day.

The USEPA used the NOAEL of 0.0008 mg/kg/day for skin and vascular lesions (Tseng et al. 1968) to derive a chronic oral RfD of 3.0E-04 mg/kg/day (IRIS 2000). The NOAEL was divided by an uncertainty factor of 3 to account for a lack of reliable data on reproductive effects and the possibility that sensitive human subgroups may not have been identified. Confidence in the RfD

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is rated medium. A higher rating was not given due to uncertainties in dose estimates and other problems in the epidemiological data base (IRIS 2000).

Carcinogenic Effects

There is strong evidence from a number of human studies that oral exposure to arsenic increases the risk of skin cancer (USEPA 1988, ATSDR 1998, NRC 1999). The most common type of cancer is squamous cell carcinoma, which appears to develop from some skin corns. In addition, basal cell carcinoma may also occur, typically arising from cells not associated with the corns. Although these cancers may be easily removed, they can be painful and disfiguring and can be fatal if left untreated. More recent data indicate that chronic oral arsenic exposure may also increase the risk of internal cancers, including cancer of the bladder and lung (NRC 1999).

The amount of arsenic ingestion that leads to skin cancer or other cancer is controversial. Based on a study of skin cancer incidence in Taiwanese residents exposed mostly to arsenic in drinking water (Tseng et al. 1968), the USEPA has calculated a unit risk of $5E-5 (\mu\text{g/L})^{-1}$ corresponding to an oral slope factor of $1.5 (\text{mg/kg/day})^{-1}$ (IRIS 2000). The NRC (1999) has reviewed a number of alternative approaches for quantification of cancer risk at low doses, and noted that the risk estimates depend heavily on the mathematical approach employed as well as the cancer data set utilized. Based on the incidence of bladder cancer in males in Taiwan, several different methods yield estimates of the EC01 (the concentration in water that results in a 1% increase in excess lifetime cancer risk) of about 400-450 $\mu\text{g/L}$. If the dose response curve is assumed to be linear and to have no threshold, this corresponds to an oral slope factor of about 0.8-0.9 $(\text{mg/kg-day})^{-1}$, generally similar to the value based on skin cancer.

Beneficial Effects

Several studies in animals suggest that low levels of arsenic in the diet may be beneficial for reproduction and normal postnatal development (ATSDR 1998). The USEPA (1988) reviewed the evidence and concluded that the essentiality of low levels of arsenic in animals has not been established, but is plausible. The NRC (1999) also reviewed the evidence and noted that studies to date do establish that arsenic supplementation of low-arsenic semi-synthetic diets prevents the occurrence of abnormal reproductive or decreased growth in animals, but that there is no proof that arsenic is an essential element in humans or that it is required for any biochemical process.

If arsenic is beneficial or essential in animals, it is also likely to be so for humans. Based on the animal data, the estimated beneficial dose for humans is approximately 10 to 50 $\mu\text{g/day}$ (USEPA 1988). This level of arsenic intake is usually provided in a normal diet, and no cases of arsenic deficiency in humans have been reported (ATSDR 1998, NRC 1999).

Summary of Toxicity Values for Arsenic

Based on the information reviewed above, this risk assessment utilized the following toxicity factors for ingested arsenic:

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Toxicity Factor	Value	Source
Chronic RfD	0.0003 mg/kg-day	IRIS 2000
Oral Slope Factor	1.5 (mg/kg-day) ⁻¹	IRIS 2000

4.3.3 Adjustments For Relative Bioavailability

As discussed in USEPA (1989), most oral RfD and SF values developed by USEPA are based on the empirical relationship between the occurrence of toxic effects and the amount of chemical ingested, and the amount of chemical that is actually absorbed into the body is not explicitly considered. Thus, if it is expected that the absorption of a chemical from an on-site medium is significantly different than from the medium used in the study supporting the RfD or SF, then it is necessary to adjust the RfD or SF to account for this difference in absorption.

The ratio of the absorption fraction for a chemical in site medium compared to the medium used in the key toxicity studies is referred to as the Relative Bioavailability (RBA). If reliable estimates of RBA are available for chemicals of potential concern in site media, these can be used to adjust the default RfD and SF values as follows:

$$RfD_{adj} = RfD_{default} / RBA$$

$$SF_{adj} = SF_{default} \cdot RBA$$

In the case of arsenic, all of the oral RfDs as well as the oral SF are based on studies of humans exposed to arsenic either in drinking water or in other readily absorbable forms. Thus, solid forms of arsenic in site soils may be less well-absorbed and require adjustments in the toxicity factors to derive appropriate estimates of toxicity.

In order to investigate the relative bioavailability of arsenic in site soils, USEPA performed a study in which five separate samples were fed to swine for 12 days. Swine were selected as the test species because it is believed the gastrointestinal system (and hence the behavior of ingested arsenic) in swine is similar to that in humans. The details of the study design and of the findings are presented in a separate report (USEPA 2000b). In brief, the study found that arsenic in site soils was less well absorbed than a readily soluble form of arsenic (sodium arsenate), with RBA values for individual samples ranging from about 0.2 to 0.5. Because it is believed that these differences in RBA reflect mainly experimental variation, a single site-wide RBA value was derived by calculating the 95% upper confidence limit of the mean RBA. The resulting value was 0.5.

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4.4 RISK CHARACTERIZATION FOR ARSENIC

4.4.1 Basic Approach

Cancer Risk

The risk of cancer from exposure to a chemical such as arsenic is described in terms of the probability that an exposed individual will develop cancer because of that exposure by age 70. For each chemical of concern, this value is calculated from the daily intake of the chemical from the site, averaged over a lifetime (DI_L), and the slope factor (SF) for the chemical, as follows (EPA 1989):

$$\text{Cancer Risk} = 1 - \exp(-DI_L \cdot SF)$$

In most cases (except when the product of $DI_L \cdot SF$ is larger than about 0.01), this equation may be accurately approximated by the following:

$$\text{Cancer Risk} = DI_L \cdot SF$$

The level of total cancer risk that is of concern is a matter of personal, community and regulatory judgement. In general, it is the policy of the USEPA that where excess cancer risks to the RME individual do not exceed a level of $1E-04$, remedial action is generally not warranted (USEPA 1991b).

Noncancer Risk

The potential for noncancer effects from exposure to a chemical is evaluated by comparing the estimated daily intake of the chemical over a specific time period with the RfD for that chemical derived for a similar exposed period. This comparison results in a noncancer Hazard Quotient, as follows (EPA 1989a):

$$HQ = DI / RfD$$

where:

HQ = Hazard Quotient

DI = Daily Intake (mg/kg-day)

RfD = Reference Dose (mg/kg-day)

If the HQ for a chemical is equal to or less than one ($1E+00$), it is believed that there is no appreciable risk that noncancer health effects will occur. If an HQ exceeds $1E+00$, there is some possibility that noncancer effects may occur, although an HQ above $1E+00$ does not indicate an effect will definitely occur. This is because of the margin of safety inherent in the derivation of all RfD values. However, the larger the HQ value, the more likely it is that an adverse effect may occur.

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As noted earlier, noncancer risks from lead are evaluated using a somewhat different approach that evaluates the likelihood of an exposed person having a blood lead level that exceeds a level of potential health concern. This evaluation of noncancer risks from lead is presented in Section 5.

4.4.2 Risks from Soil and Dust

Cancer Risk

Cancer risks from exposure of residents to arsenic in yard soil and indoor house dust were calculated for each property using the basic equations described above. The concentration term used for soil at each property was the 95% UCL of the mean or the maximum value (whichever was lower), and the concentration in dust was calculated from the soil concentration as described in Section 4.2.4 (above). The resulting risk estimates are shown in Table 4-1.

For CTE exposure conditions, most properties have estimated excess cancer risks for exposures due to arsenic in soil plus dust that lie between $1.1\text{E-}06$ and $1.4\text{E-}05$ (5th to 95th percentile), with a maximum value of $8.3\text{E-}05$.

For RME exposure conditions, most properties have risk estimates that lie between $1.0\text{E-}05$ and $1.3\text{E-}04$, with a maximum value of $7.8\text{E-}04$. Of the total properties investigated (1548), 112 (approximately 7%) have RME cancer risks which exceed $1.0\text{E-}04$.

As shown in Table 4-1, the pattern of properties with arsenic contamination that exceeds a risk level of $1\text{E-}04$ is approximately uniform across the site, with a frequency of about 7%-9% in Cole, Clayton, and Swansea/Elyria. This approximately uniform pattern is also shown in Figure 4-1, where the site is divided into six rectangles of generally similar area, with the following frequency:

Area	N	N > $1.0\text{E-}04$	%
1	26	0	0
2	13	0	0
3	67	6	9%
4	171	11	6%
5	674	49	7%
6	600	46	8%
All	1551	112	8%

The apparent absence of properties with an RME cancer risk above $1\text{E-}04$ in Five Points and Globeville may be a consequence of the fact that only a small number of homes were sampled in these areas, rather than an authentic absence of impacted properties.

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Chronic Noncancer Risks

Estimated risks of non-cancer health effects from chronic exposure to arsenic in soil and dust are shown in Table 4-2. For individuals with CTE exposure, risks at most properties fall between $1.9\text{E-}02$ and $2.3\text{E-}01$ (5th to 95th percentile), while individuals with RME exposure have risks that lie mainly between $5.3\text{E-}02$ and $6.6\text{E-}01$. These results indicate that risk of noncancer effects is low for most individuals at most locations. However, there is one location where the CTE non-cancer risk does slightly exceed $1.0\text{E+}00$ ($\text{HQ} = 1.4\text{E+}00$), and there are 28 locations where the RME HQ values exceed $1.0\text{E+}00$ (maximum = $4.0\text{E+}00$). These locations where noncancer risks enter a range of concern ($\text{HQ} > 1\text{E+}00$) are also above the usual level of concern ($1\text{E-}04$) for cancer.

Noncancer Risks from Short-Term Exposures

In most cases, if chronic noncancer and cancer risks from arsenic are below a level of concern, risks from shorter term exposures will also be below a level of concern. However, in cases where there is high spatial variability of arsenic concentrations in soil at different locations within a property (e.g., see Figure 2-3), it is conceivable that long-term average exposures in a yard might not be of concern, but that short-term high-intake exposures by a child playing in the yard at locations of above-average concentration could be of concern. This is commonly referred to as a "hot-spot" exposure scenario.

The basic equation used to evaluate noncancer risk from this type of scenario is the same as described previously:

$$\text{HQ} = C \cdot (\text{IR}/\text{BW} \cdot \text{EF} \cdot \text{ED}/\text{AT}) / (\text{RfD}/\text{RBA})$$

However, the values of the inputs to the equation are modified to reflect the short-term nature of the exposure. Each of the inputs are discussed below.

Concentration

As before, the concentration term needed to assess short-term exposure is the mean concentration averaged over the location where the exposure takes place. For longer term exposure, the area over which averaging occurs is the entire yard. However, for short-term exposure, the area in which exposure occurs could be smaller, depending on the length of time involved. In the absence of any specific data on short-term exposure patterns by children, it is assumed this area is about 1/10 that of the entire yard.

Because the soil samples collected during Phase III were 10-point composites rather than grab samples, these data are not suited for direct estimation of the average concentration over an area 1/10 of the yard. However, it is possible to estimate the maximum concentration which could theoretically exist at any subarea of the yard by assuming that 9 of the 10 sub-samples were from locations where the soil concentration was at background levels, and the tenth sub-sample was from a "hot-spot". In this case, the concentration in the composite would be:

$$C(\text{composite}) = (9 \cdot \text{Bkg} + 1 \cdot \text{Hotspot}) / 10$$

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Thus, given the concentration value for a composite, the highest concentration that any sub-sample in that composite could possibly have is:

$$C_{\max}(\text{hotspot}) = 10 \cdot \text{Composite} - 9 \cdot \text{Bkg}$$

The value of the mean background concentration of arsenic at the site is not established with certainty, but based on the distribution of data collected during the Phase III sampling program, it seems likely the value is about 15-20 ppm. Assuming an intermediate value of 17 ppm, the maximum theoretical hotspot concentration (MTHC) is given by:

$$\text{MTHC} = 10 \cdot \text{Composite} - 150 \text{ ppm}$$

The MTHC was calculated for each property based on the maximum composite value from each property, and this value was used as a screening-level input to the short-term risk equation. If the calculated HQ based on the MTHC does not exceed 1E+00, it can reliably be concluded that short term risks are not of concern. However, it is essential to note that if the calculated HQ based on the MTHC does exceed 1E+00, this does not establish that there is a risk. Rather, this result indicates that a short-term risk might exist, and that additional investigation is needed to assess this possibility.

Body Weight

Because the scenario of chief concern is a small child playing in the yard, the body weight was assumed to be 10 kg. This corresponds to a child who is 6-12 months in age.

Soil Intake Rate

The average amount of soil ingested per day by a child during a short-time exposure is not known. USEPA typically assumes that an intake of about 200 mg/day is the upper bound for the average intake across a time interval of 1 year. In the absence of data, it was assumed that the average intake over a period of several months (sub-chronic) or several days (sub-acute) was 500 mg/day and 2,000 mg/day, respectively.

Exposure Frequency

The short-term frequency with which a small child will be in direct contact with yard soil is not known. For sub-chronic exposure, it is assumed that the highest exposure frequency will occur in the warm summer months (120 days), and that the child will play in the yard about 30 of those days. For sub-acute exposure, it is assumed the child is exposed in the yard one day out of two.

Toxicity Factors

The USEPA has developed a subchronic oral RfD for arsenic of 6E-03 mg/kg-d (USEPA 1995b). This value is based on an estimated LOAEL of 0.06 mg/kg-day in humans (both children and

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adults) exposed to arsenic for periods of time from six months up to about 15 years. An uncertainty factor of 10 is used to account for extrapolation from a LOAEL to a NOAEL.

The USEPA has not yet developed any acute or sub-acute RfD values for arsenic. Such a factor is needed to assess exposures of several days to several weeks in duration. However, observation of short-term toxicity in human have not been noted for exposures lower than about 0.05 mg/kg-day (ATSDR 1998):

Study	Number of patients	Exposure Duration	LOAEL mg/kg-day
Armstrong et al. 1984	5	1 week	1
Wagner et al. 1979	1	4 mo.	0.05
Cullen et al. 1995	1	1 dose	17
Franzblau and Lillis 1989	2	1-2 mo.	0.29
Mizuta et al. 1956	220	2-3 wk	0.05

In the absence of any USEPA or ATSDR-validated values for sub-acute exposure, a value of 0.05 mg/kg-day will be utilized in this assessment for estimating risks from sub-acute exposure. No reliable estimate of an acute (single dose) RfD is available.

Results

Based on the exposure assumptions above, and using the MTHC as the input concentration, the following results are obtained:

Result	Sub-Acute	Sub-Chronic
HQ < 1.0E+00 (no concern over hot spots)	1387	1382
HQ > 1.0E+00 (possible hot spot)	161	166
HQ > 1.0E+00 AND cancer risk < 1.0E-04	49	54

As seen, about 161-166 properties are identified by this screening technique as locations where a hot-spot of potential short-term noncancer health concern might exist. Of these properties, most are already identified as being of potential concern due to estimated chronic cancer risks which exceed a cancer risk level of 1.0E-04. The remaining properties (about 49-54) are locations where additional investigation may be needed to determine if any short-term risks are actually present.

4.4.3 Risks from Home-Grown Vegetables

A total of 72 different samples of garden vegetables were collected from 19 different properties across the site. At each property, the 95% UCL of the mean concentration of arsenic was calculated, and this value (or the maximum, whichever was lower) was used to estimate risks to residents. The results are summarized in Table 4-3.

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As seen, for individuals whose intake of home-grown garden vegetables is average (CTE) for the western United States, neither non-cancer nor cancer risks enter a range of concern at any property tested. For individuals whose intake is at the upper-bound (RME) of the distribution of garden vegetable consumption, risks do enter a range of potential concern for five properties. At three of these properties (numbers 4, 8 and 12), the magnitude of the excess risk is relatively small (RME cancer risk = $2E-04$), and is due in most cases to the conservatism introduced by use of the 95% UCL of the mean rather than the mean concentration for the risk calculations.

At property 11, the high risk estimates are attributable to a single vegetable sample (garlic) that was significantly higher than the remainder of the samples from this location. Because this value seemed to be questionable compared to other samples from the garden, EPA returned to the property and collected a second sample of garlic. This sample yielded a much lower concentration for arsenic (0.2 ppm vs 1.24 ppm), suggesting the first result may have been anomalous. In any event, because it is likely that the mass of garlic ingested per day is relatively small, risks at this location are not likely to be of concern.

At property 6, a number of vegetables had concentration values that were higher than in samples from most other properties, and samples from this location could be of potential concern for an RME consumer. The concentrations of arsenic in the garden soil samples at this location were also somewhat higher (mean = 51 ppm) than for most other gardens (average = 15 ppm, range = 11 to 24 ppm), suggesting the elevated values were attributable to soil contamination. An interview with the property owner did not reveal any probable source of arsenic in the garden.

An alternative approach for evaluating the risks from arsenic in garden vegetables is to compare the doses calculated from site vegetable data to those that occur through the normal diet. These data are summarized below:

Parameter	Value (ug/kg-day)
Typical dietary intake of arsenic ^a	0.36 - 0.81
Estimated Intake at VBI70 Properties	
Property 6	0.26 - 2.63
Property 11	0.10 - 0.99
All other properties	0.001 - 0.35

^a Gunderson 1995

As seen, intakes of arsenic from vegetables are generally within the typical amount in the diet at all locations except for Property 6, supporting the conclusion that neither intakes nor risks are excessive at locations where garden soils do not exceed about 50 ppm.

4.5 UNCERTAINTIES IN ARSENIC RISK ASSESSMENT

It is important to recognize that the calculations of short-term and long term exposure and risk from arsenic ingestion in soil are based on a number of assumptions and estimates, and that these

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introduce uncertainty into the risk results. The most important of the sources of uncertainty in the calculations are summarized below.

Uncertainty in Concentration Terms

The concentration term that is appropriate for calculating exposure and risk from ingestion exposure to arsenic is the true mean concentration in the medium of concern (soil, dust, vegetables), averaged over the area and time interval (averaging time) of concern. Because the true mean cannot be calculated from a limited set of sample results, the USEPA utilizes the 95% upper confidence limit of the mean as a conservative estimate of the true mean. This approach helps ensure that the exposure and risk estimates that are derived are more likely to overestimate than underestimate the actual risk.

On the other hand, data on the relation between the concentration of arsenic in bulk (unsieved) soil samples and in the fine (sieved) fraction indicates arsenic may tend to be somewhat more concentrated (perhaps about 20%) in the fines than in the bulk. USEPA generally assumes that when soil is ingested by hand to mouth contact, it is likely that the particles ingested will be mainly fines. If this is true, exposure calculations based on the measured concentration in bulk samples may tend to underestimate risk by about 20%.

Uncertainty in Intake Rates

Data on the amount of soil ingested by humans are very limited. Measurements are difficult to perform, and results vary significantly from study to study and from method to method. In addition, data are based mainly on short term studies, so estimates of long-term average intake rates are especially uncertain. Moreover, intake rates are likely to vary from site to site and property to property, depending on things such as climate, socioeconomic status, yard condition, etc, so the default intake rates used in these calculations may not reflect the true intake rates at the site. Because of the limitations in the data, the default values recommended by USEPA are intended to be on the high side (i.e., are more likely to overestimate than underestimate actual soil ingestion).

Uncertainty in Exposure Duration

Cancer risk calculations depend on the duration of exposure. Default exposure durations used in the risk assessment are not site-specific, and are estimated from data on the length of time that people own a particular residence. Thus, actual exposure durations of residents at the site may not be the same as the assumed exposure durations assumed, and might be either longer or shorter than assumed.

Uncertainty in Toxicity Factors

One of the largest sources of uncertainty in most risk assessments stems from uncertainty in the toxicity factors used to predict responses from the calculated doses. In the case of arsenic, dose-response data are derived from studies in humans, which significantly reduces the degree of uncertainty compared to extrapolations based on animal data. However, a significant degree of

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uncertainty still remains in all of the toxicity factors. For example, dose-response curves in the key studies are generally limited by lack of precise data on the actual exposure rates. Moreover, there are still large uncertainties in how to extrapolate from relatively high exposure levels to lower exposure levels, and in the importance of cultural and ethnic differences between different study populations. USEPA is currently in the process of reassessing the risk characterization for arsenic, and the quantitative risk factors (RfD and/or oral slope factor) may be revised in the future as new data and as new analyses warrant.

Uncertainty in Bioavailability

In order to cause an adverse response, arsenic that is ingested must be absorbed into the body. As detailed in USEPA (2000b), measurements of the arsenic relative bioavailability have been performed for five soils from the VBI70 site. While measurements based on site soils significantly reduces uncertainty in this exposure parameter, uncertainty still remains. For example, variability was observed between different site soils, and a conservative estimate of the mean value was employed to represent the site-wide average absorption. This approach is expected to result in an over-estimate of true absorption. Another source of uncertainty is in the extrapolation of data from test animals to humans. The test animals (swine) were selected because they are believed to have a gastrointestinal system similar to that in humans, but it is also expected that absorption in humans may vary as a function of age, stomach contents, nutritional status, etc. Thus, the measurements in animals should be viewed as uncertain estimates of the true values in humans.

Uncertainty in Short-Term Exposure and Risks

As noted earlier, short-term exposures to arsenic might be of concern at some properties (see Section 4.4.2), depending on the actual short-term level of soil ingestion by a child, and on the size of the area over which a child might be exposed during short term activities. For the purposes of the calculations, an area 1/10 the size of the yard was assumed. This is probably reasonable for sub-acute exposures (those which occur over the course of several days), but might be somewhat conservative for sub-chronic exposures (those which occur over the course of several months). Because no data are currently available to improve on the estimates of short-term exposure from soil (either the actual intake rates or the area over which exposure occurs), the most effective approach for evaluating this type of risk would be an on-going program of biomonitoring in community residents, especially children. Such a program would provide information on the frequency and level of non-dietary arsenic exposures that may be of either short-term or long term health concern.

Summary

Because of the uncertainties summarized above, none of the exposure and risk calculations for arsenic should be interpreted as accurate measures of the true risk. Rather, all values should be interpreted as uncertain estimates. Because most of the approaches for dealing with uncertainty are intended to be conservative (i.e., are more likely to overestimate than underestimate), the risk values above should be thought of as high-end estimates of the true risk, and actual risks are likely to be lower.

TABLE 4-1 ESTIMATED CANCER RISK FROM ARSENIC IN SOIL AND DUST

Neighborhood	Properties Evaluated	CTE Cancer Risk				RME Cancer Risk			
		≤ 1E-05	1E-05 - 1E-04	1E-04 - 1E-03	> 1E-03	≤ 1E-05	1E-05 - 1E-04	1E-04 - 1E-03	> 1E-03
Clayton	644	587	57	0	0	219	371	54	0
		91.1%	8.9%	0.0%	0.0%	34.0%	57.6%	8.4%	0.0%
Cole	601	554	47	0	0	124	437	40	0
		92.2%	7.8%	0.0%	0.0%	20.6%	72.7%	6.7%	0.0%
Five Points	14	14	0	0	0	2	12	0	0
		100.0%	0.0%	0.0%	0.0%	14.3%	85.7%	0.0%	0.0%
Globeville	22	22	0	0	0	4	18	0	0
		100.0%	0.0%	0.0%	0.0%	18.2%	81.8%	0.0%	0.0%
Swansea/Elyria	267	247	20	0	0	92	157	18	0
		92.5%	7.5%	0.0%	0.0%	34.5%	58.8%	6.7%	0.0%
All Neighborhoods	1548	1424	124	0	0	441	995	112	0
		92.0%	8.0%	0.0%	0.0%	28.5%	64.3%	7.2%	0.0%

TABLE 4-2 ESTIMATED CHRONIC NONCANCER RISK FROM ARSENIC IN SOIL AND DUST

Neighborhood	Properties Evaluated	CTE Hazard Quotient					RME Hazard Quotient				
		≤1	1-2	2-5	6-10	> 10	≤1	1-2	2-5	6-10	> 10
Clayton	644	643	1	0	0	0	633	7	4	0	0
		99.8%	0.2%	0.0%	0.0%	0.0%	98.3%	1.1%	0.6%	0.0%	0.0%
Cole	601	601	0	0	0	0	585	10	6	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	97.3%	1.7%	1.0%	0.0%	0.0%
Five Points	14	14	0	0	0	0	14	0	0	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	100.0%	0.0%	0.0%	0.0%	0.0%
Globeville	22	22	0	0	0	0	22	0	0	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	100.0%	0.0%	0.0%	0.0%	0.0%
Swansea/Elyria	267	267	0	0	0	0	266	1	0	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	99.6%	0.4%	0.0%	0.0%	0.0%
All Neighborhoods	1548	1547	1	0	0	0	1520	18	10	0	0
		99.9%	0.1%	0.0%	0.0%	0.0%	98.2%	1.2%	0.6%	0.0%	0.0%

TABLE 4-3 ESTIMATED RISK FROM ARSENIC IN GARDEN VEGETABLES

Property Number	Neighborhood	Sample Size	Concentration in Vegetable (mg/kg ww)			Chronic Noncancer Risk		Lifetime Cancer Risk	
			Mean	Max	EPC(a)	CTE	RME	CTE	RME
1	CLAYTON	10	3.3E-03	8.1E-03	5.3E-03	8E-03	9E-02	5E-07	2E-05
2	CLAYTON	1	4.1E-03	4.1E-03	4.1E-03	6E-03	7E-02	4E-07	1E-05
3	CLAYTON	1	4.4E-02	4.4E-02	4.4E-02	7E-02	7E-01	4E-06	1E-04
4	CLAYTON	6	1.8E-02	5.5E-02	5.5E-02	9E-02	9E-01	5E-06	2E-04
5	CLAYTON	2	1.2E-02	1.9E-02	1.9E-02	3E-02	3E-01	2E-06	6E-05
6	CLAYTON	12	1.7E-01	9.8E-01	5.4E-01	9E-01	9E+00	5E-05	2E-03
7	CLAYTON	2	1.0E-02	1.6E-02	1.6E-02	3E-02	3E-01	1E-06	5E-05
8	COLE	2	5.2E-02	6.7E-02	6.7E-02	1E-01	1E+00	6E-06	2E-04
9	COLE	2	1.7E-03	1.8E-03	1.8E-03	3E-03	3E-02	2E-07	5E-06
10	COLE	1	1.9E-03	1.9E-03	1.9E-03	3E-03	3E-02	2E-07	6E-06
11	COLE	6	5.0E-02	2.0E-01	2.0E-01	3E-01	3E+00	2E-05	6E-04
12	COLE	4	4.4E-02	7.3E-02	7.3E-02	1E-01	1E+00	7E-06	2E-04
13	COLE	9	1.3E-02	3.4E-02	3.4E-02	5E-02	5E-01	3E-06	1E-04
14	COLE	3	1.1E-02	1.9E-02	1.9E-02	3E-02	3E-01	2E-06	6E-05
15	COLE	4	1.0E-02	3.1E-02	3.1E-02	5E-02	5E-01	3E-06	1E-04
16	COLE	1	2.0E-02	2.0E-02	2.0E-02	3E-02	3E-01	2E-06	6E-05
17	SWANSEA/ELYRIA	2	2.4E-03	3.4E-03	3.4E-03	5E-03	5E-02	3E-07	1E-05
18	SWANSEA/ELYRIA	1	1.4E-03	1.4E-03	1.4E-03	2E-03	2E-02	1E-07	4E-06
19	SWANSEA/ELYRIA	3	2.6E-03	4.8E-03	4.8E-03	7E-03	8E-02	4E-07	1E-05

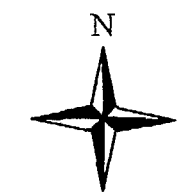
Notes:

Shading indicates that vegetable concentration and resulting risk may exceed protective levels.

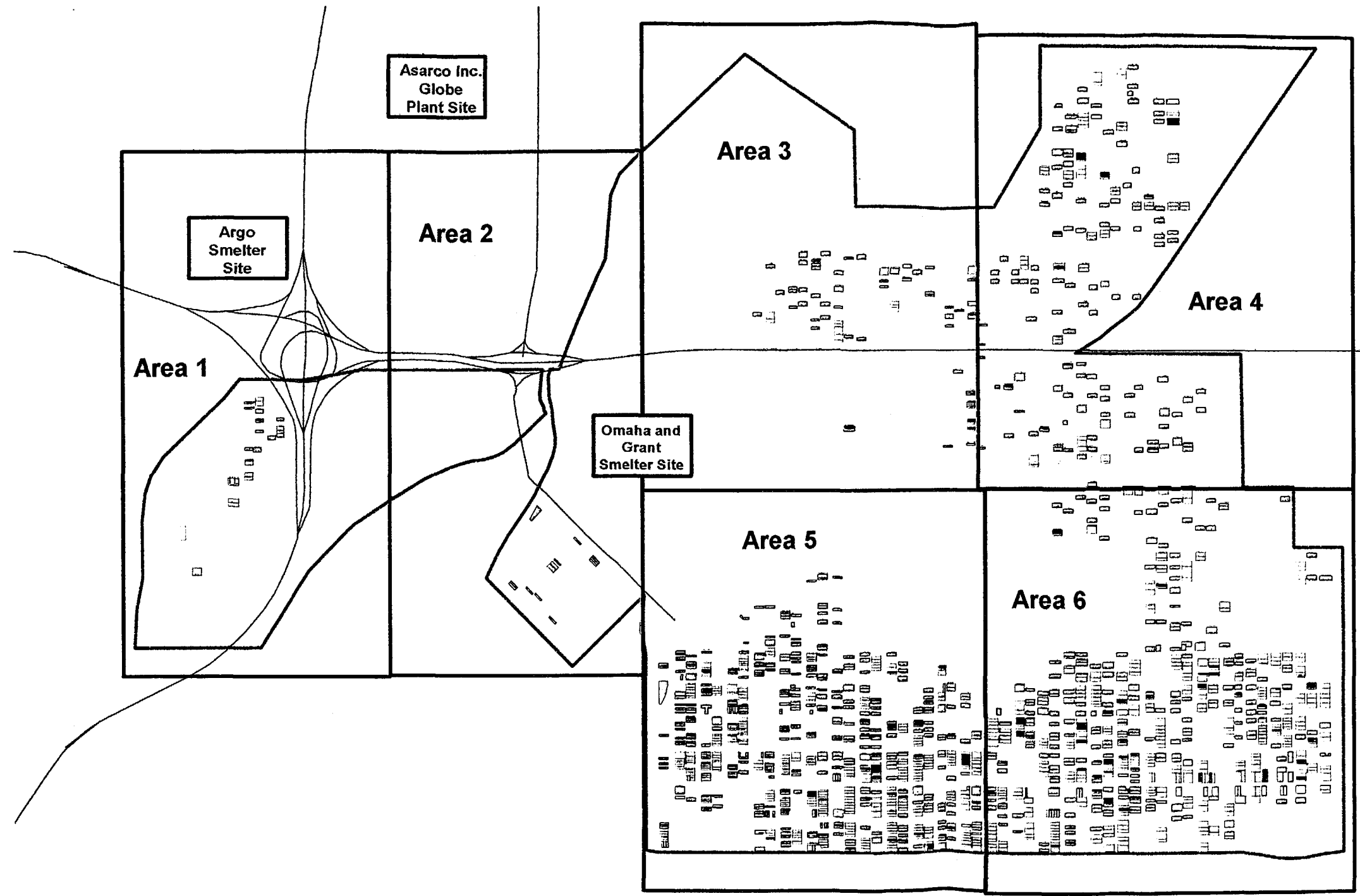
(a) EPC = 95% UCL or max

Figure 4-1

**Map of Properties
with RME Cancer Risks
Above 1E-04**



Arsenic EPC (ppm)
◻ ≤ 1E-04
◼ > 1E-04



SECTION 5

EXPOSURE AND RISK FROM LEAD

5.1 OVERVIEW

As noted earlier, risks from lead are evaluated using a somewhat different approach than for most other metals. First, because lead is wide-spread in the environment, exposure can occur by many different pathways. Thus, lead risks are usually based on consideration of total exposure (all pathways) rather than just to site-related exposures. Second, because studies of lead exposures and resultant health effects in humans have traditionally been described in terms of blood lead level (PbB, expressed in units of ug/dL), lead exposures and risks are typically assessed using an uptake-biokinetic model rather than an RfD approach. Therefore, calculating the level of exposure and risk from lead in soil also requires assumptions about the level of lead in other media, and also requires use of pharmacokinetic parameters and assumptions that are not needed in traditional methods.

For residential land use, the sub-population of chief concern is young children. This is because young children 1) tend to have higher exposures to lead in soil, dust and paint, 2) tend to have a higher absorption fraction for ingested lead, and 3) are more sensitive to the toxic effects of lead than are older children or adults.

It is currently difficult to identify what degree of lead exposure, if any, can be considered safe in young children. Some studies report subtle signs of lead-induced neurobehavioral effects in children beginning at around 10 ug/dL or even lower, with population effects becoming clearer and more definite in the range of 30-40 ug/dL (ATSDR 1998, CDC 1991). On the other hand, some researchers and clinicians believe the effects that occur in children at low blood lead levels are so minor that they need not be cause for concern.

After a thorough review of all the data, the USEPA has identified 10 ug/dL as the blood lead level at which effects that warrant avoidance begin to occur, and has set as a goal that there should be no more than a 5% chance that any child will have a blood lead value above 10 ug/dL (EPA 1994a, 1994b). This approach focuses on the risks to a child at the upper bound (about the 95th percentile) of the exposure distribution, very much the same way that the approach used for other chemicals focuses on risks to the RME individual. The Centers for Disease Control (CDC) has also established a guideline of 10 ug/dL in preschool children which is believed to prevent or minimize lead-associated cognitive deficits (CDC 1991).

5.2 IEUBK MODEL FOR ASSESSING LEAD RISK

The USEPA has developed an Integrated Exposure Uptake Biokinetic (IEUBK) model for predicting the likely range of blood lead levels in a population of young children (age 0-6 years) exposed to a specified set of environmental lead levels (USEPA 1994b). This model requires as input data on the levels of lead in soil, dust, water, air, and diet at a particular location, and on the amount of these media ingested or inhaled by a child living at that location. All of these inputs to

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the IEUBK model are central tendency point estimates. These point estimates are used to calculate an estimate of the central tendency (the geometric mean) of the distribution of blood lead values that might occur in a population of children exposed to the specified conditions. Assuming the distribution is lognormal, and given (as input) an estimate of the variability between different children (this is specified by the geometric standard deviation or GSD), the model calculates the expected distribution of blood lead values, and estimates the probability that any random child might have a blood lead value over 10 ug/dL.

If all of the IEUBK model exposure levels and intake rates are set at their default values, and if the concentration of lead in dust is assumed to be 70% of that in soil (the default assumption), then the IEUBK model predicts that a soil lead level of about 350 ppm corresponds to the target risk level (no more than a 5% chance of exceeding a blood lead level of 10 ug/dL). If default estimates of dietary intake are adjusted downwards by a factor of 0.7 to reflect lower lead levels in the current food supply (Bolger et al. 1996, Gunderson et al. 1995, Griffin et al. 1999) than are assumed in the default IEUBK model, then the soil lead level that corresponds to the target risk level is about 400 ppm. Based in part on these results, USEPA has established a national policy that soil lead levels below 400 ppm may be assumed to be below a level of health concern (EPA 1994a). Soil lead levels above 400 ppm may or may not be of concern, depending on site-specific factors.

Whenever reliable site-specific data are available on any of the IEUBK model input parameters, these are used in preference to the assumptions employed in the default case. At this site, the only site-specific information available for refining the estimate of lead risk to children is the measured relationship between lead in soil and lead in indoor dust (see Figure 2-8):

$$C_d = 0.34 \cdot C_s + 150$$

As shown in Figure 5-1, when this site-specific soil-dust relationship is employed in the IEUBK model in place of the default assumption ($C_d = 0.7 \cdot C_s$), the soil lead concentration that corresponds to the target risk level does not change significantly, but the predicted blood lead values at soil lead concentrations above 400 ppm are somewhat lower than those predicted by the default model. These adjustments to the model, along with the other model inputs, are summarized in Table 5-1. This site-specific adjusted model was used to evaluate risks to children from lead in soil and dust, as described below.

5.3 RISK CHARACTERIZATION FOR LEAD

5.3.1 Risks from Lead in Soil and Dust

The risk to children (age 0-84 months) was calculated for each property, using the mean soil lead concentration as input, and predicting the dust lead concentration using the equation above. The results are shown in Table 5-2.

As seen, of the total of 1,548 residences examined, a total of 159 (10%) have mean soil lead levels above 400 ppm. In most of these cases the soil lead is only slightly elevated, with 137 of the 159 being less than 600 ppm. When characterized in terms of predicted risk of exceeding a blood lead

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level of 10 ug/dL (this is referred to as "P10"), the majority of homes (108 out of 159) above the 400 ppm soil lead level would be expected to have P10 values of 5-10%, only slightly above the health-based goal. However, about 40 properties would be expected to have P10 values between 10-20%, and 11 homes are predicted to have P10 values greater than 20%.

Although homes with elevated soil lead are found in all neighborhoods, the density of homes above 400 ppm tends to be higher in the central and western part of the site than in areas on the eastern side of the site. This is illustrated in Figure 5-2 and in the following table:

Area	N	N > 400 ppm	%
1	26	2	8%
2	13	6	46%
3	67	25	37%
4	171	5	3%
5	674	107	16%
6	600	14	2%

5.3.2 Risks from Lead in Garden Vegetables

As shown previously (see Figure 2-9), there is essentially no uptake of lead from soil into garden vegetables at this site. On this basis, it is concluded that exposure to lead from ingestion of home grown garden vegetables is not of concern.

5.4 UNCERTAINTIES IN LEAD RISK EVALUATION

It is important to stress that lead risk predictions based on the IEUBK model are uncertain. This uncertainty arises from a number of factors. First, there is inherent difficulty in providing the model with reliable estimates of human exposure to lead-contaminated media. For example, exposure to soil and dust is difficult to quantify because human intake of these media is likely to be highly variable, and it is very difficult to derive accurate measurements of actual intake rates. Likewise, site-specific data on exposure to lead through the diet is generally not available, and because dietary lead levels have been decreasing over time, the default data used in the model may no longer be accurate. Second, it is often difficult to obtain reliable estimates of key pharmacokinetic parameters in humans (e.g., absorption fraction, distribution and clearance rates, etc.), since direct observations in humans are limited. Finally, the absorption, distribution and clearance of lead in the human body is an extremely complicated process, and any mathematical model intended to simulate the actual processes is likely to be an over-simplification. Consequently, IEUBK model calculations and predictions should not be thought of as being identical to actual risk.

One way to help determine whether the IEUBK model is yielding reliable results at a particular site is to compare the IEUBK model predictions with actual observations of blood lead levels in the population of children currently living at the site. This approach has been used at a number of other sites in the Rocky Mountain west (e.g., Aspen, Leadville, Midvale), and it is usually found

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that the observed incidence of elevated blood lead values is not as high as predicted by the model. There are a number of reasons why this might be so, including potential limitations in the blood lead study itself. However, the consistency of this pattern across sites suggests that, on average, the default IEUBK model may tend to be somewhat over-conservative. If so, this would presumably stem from imprecision in one or more of the model inputs (especially in intake rates and biokinetic factors), but the actual basis source of the apparent discrepancies between predicted and observed blood lead values remains uncertain and controversial.

At the VBI70 site, only very limited blood lead data are available. These data were derived from a study of individuals living at homes selected for soil removal as part of the Phase II program. These individuals were asked to allow sampling of hair and urine to assess arsenic exposure, and sampling of blood to assess lead exposure. A total of 15 individuals participated. The results for blood lead are summarized below:

Age category	N	Lead in Soil (ppm)		PbB (ug/dL)	
		Mean	Max	Geometric mean	Max
Child (0-7 years)	2	426	426	2.0	2.0
Adult (>7 Years)	13	489	1318	2.1	4.0
All	15	484	1318	2.2	4.0

This data set is much too limited to support any strong conclusion, especially because the number of children participating was so low, and because many of the properties had lead levels in soil that were only moderately elevated. However, the data do not provide a basis for concluding that lead exposures are above a level of concern.

Because of the potential uncertainties that may be associated with the use of the IEUBK model in selecting an action level for soil in residential areas, it is sometimes useful to consider the results of alternative approaches. Perhaps the most direct approach is to investigate the efficacy of soil remediation in reducing blood lead values in residential children. A study of this sort has been performed by the USEPA in urban areas of Baltimore, Boston and Cincinnati (EPA 1995a). Because of this study design, this investigation is usually referred to as the "three cities study". Among the key findings of this study was the conclusion that:

"...soil abatement alone will have little or no effect on reducing exposure to lead unless there is a substantial amount of lead in soil and unless this lead is the primary source of lead in house dust"

The report did not rigorously define "substantial", but it was only when soil lead levels were higher than 1,000-2,000 ppm that a benefit from soil remediation was detectable. Conversely, in two cities where soil lead levels were mainly less than 1,000 ppm, no substantial decrease in blood leads could be detected following soil remediation. As noted earlier, 99% of all properties tested in Phase III have soil lead concentrations below 700 ppm, with only one property being above 1,000 ppm (1,131 ppm). Also recall that, at the VBI70 site, available data indicate that

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only about 34% of the mass of interior dust appears to be derived from yard soil. Thus, it appears that neither of the two conditions needed for soil removal to be effective are likely to apply at most properties at the VBI70 site.

Another approach for assessing hazard from lead in soil is currently under development by USEPA Region VIII. This approach, referred to as the Integrated Stochastic Exposure (ISE) Model for Lead, is similar to the IEUBK model, except that it uses probability density functions (PDFs) rather than point estimates as inputs for most concentration and exposure parameters. These distributions are combined using Monte Carlo simulation techniques to yield a predicted distribution of absorbed lead doses (ug/day) for different members of the exposed population. These doses are then used as input to the biokinetic portion of the IEUBK model in order to generate the predicted distribution of blood lead values in the population. Thus, the variability between children is evaluated in the ISE model based on the variability in environmental and exposure parameters, rather than by application of an assumed or estimated GSD value as in the IEUBK model. A more complete description of the model and of the input parameters can be found in Goodrum et al. (1996). Because this model has not yet undergone peer review or validation, it is considered to be only an investigative tool. However, when the ISE model is used to estimate the relation between soil lead and blood lead using the inputs listed in Table 5-3, the probability of a child having a blood lead value higher than 10 ug/dL does not exceed the health-based goal of 5% until soil lead levels exceed about 1,500 ppm.. Based on the results from Phase 3a soil sampling, no properties were located where mean soil lead levels exceed 1,500 ppm, suggesting lead risks to children might not be of concern at this site.

Although none of these alternative approaches form a sufficient basis for selecting an alternative soil action level for lead, they do provide a sense as to uncertainty in the value, and the range of alternative values that might be sufficient to ensure protection of public health.

TABLE 5-1 IEUBK MODEL INPUTS

SOIL/DUST INPUTS

Csoil = property-specific average (ppm)

Cdust = 0.34*Csoil + 150 (ppm)^a

CONSTANTS

PARAMETER	VALUE
Air concentration (ug/m ³)	0.10
Indoor air concentration	30% of outdoors
Drinking water concentration (ug/L)	4.0
Absorption Fractions:	
Air	32%
Diet	50%
Water	50%
Soil/Dust	30%
Other ^a	30%
Fraction soil	45%
GSD	1.6

AGE DEPENDENT

Age	AIR		DIET	WATER	SOIL	OTHER ^a
	Time Outdoors (hrs)	Vent. Rate (m ³ /day)	Dietary intake (ug/day)	Intake (L/day)	Intake (mg/day)	Intake (ug/day)
0-1	1.0	2.0	3.87	0.20	85	5.74
1-2	2.0	3.0	4.05	0.50	135	9.11
2-3	3.0	5.0	4.54	0.52	135	9.11
3-4	4.0	5.0	4.37	0.53	135	9.11
4-5	4.0	5.0	4.21	0.55	100	6.75
5-6	4.0	7.0	4.44	0.58	90	6.08
6-7	4.0	7.0	4.90	0.59	80	5.74

^a The baseline concentration of lead in dust (11 ppm) is entered using the OTHER input menu

TABLE 5-2 ESTIMATED RISKS TO CHILDREN FROM LEAD IN SOIL AND DUST

Neighborhood	Count	Count >400	P10 >=5 and <10	P10 >=10 and <20	P10 >=20
CLAYTON	644	18	11	4	3
	100%	2.8%	1.7%	0.6%	0.5%
COLE	601	104	77	23	4
	100%	17.3%	12.8%	3.8%	0.7%
FIVE POINTS	14	7	3	3	1
	100%	50.0%	21.4%	21.4%	7.1%
GLOBEVILLE	22	2	1	0	1
	100%	9.1%	4.5%	0.0%	4.5%
SWANSEA/ELYRIA	267	28	16	10	2
	100%	10.5%	6.0%	3.7%	0.7%
ALL	1548	159	108	40	11

TABLE 5-3 ISE MODEL INPUTS

Soil-dust Relationship

$$\text{Dust} = 150 + 0.34 * \text{Soil}$$

Intake Rate Parameters

Parameter	Description (units)	PDF	Parameters
IR _{sd}	Soil/dust ingestion rate (mg/day)	PDF cumulative	{0, 10, 45, 88, 186, 208, 225, 7000} {0, 0.25, 0.50, 0.75, 0.90, 0.95, 0.99, 1.0}
WF _{age}	Weighting factor, age (unitless)	Point estimate	Same as IEUBK
WF _{soil}	Weighting factor, soil (unitless)	Triangular	{min, mode max} = {0.30, 0.45, 0.60}

Exposure Frequency

Parameter	Description (units)	PDF	Parameters
EF	Exposure Frequency (days)	Triangular	{200, 234, 350} {min, mode, max}

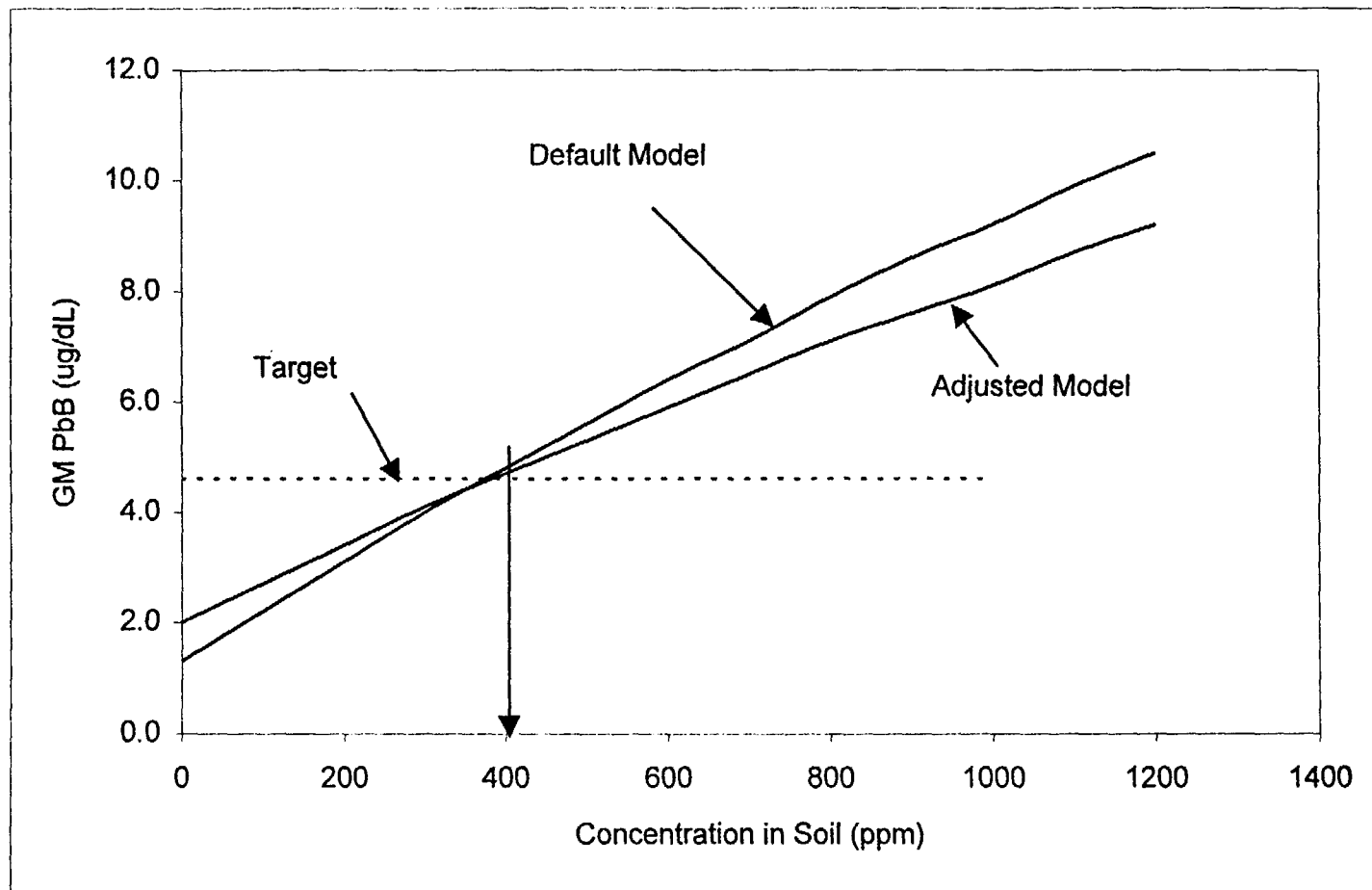
Other Parameters

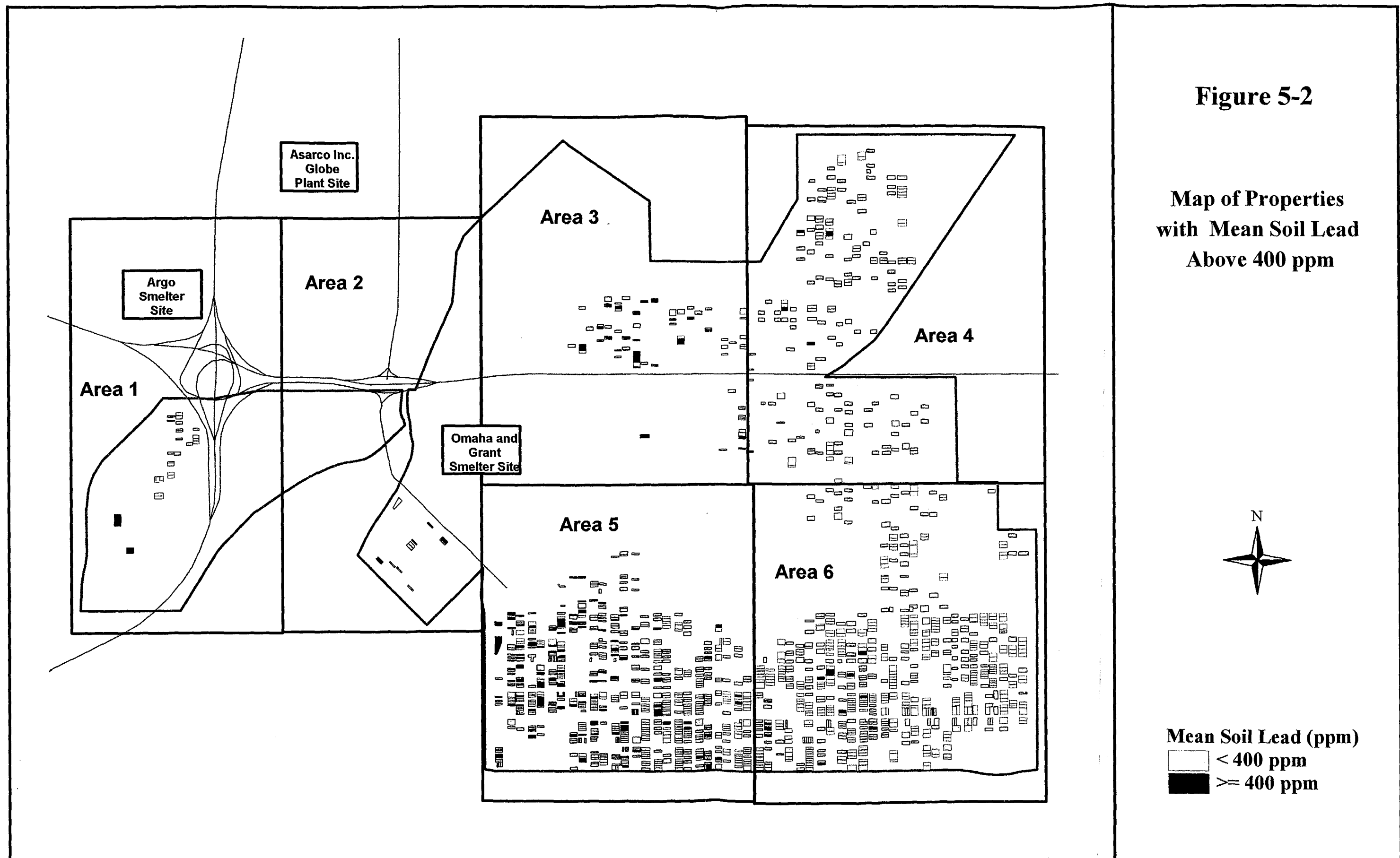
$$\text{RBA} = 0.60 \text{ (Point estimate)}$$

Concentration of lead in water and air (point estimates) – The concentration of water was estimated as 4 ug/L. For air, a default value of 0.1 ug Pb/m³ air was used.

Other values that were entered as point estimates include air inhalation rates, absorption fractions of water, diet, and other (non-soil/dust). These values were left as parameters provided in the ISE model (Goodrum et al. 1996).

FIGURE 5-1 DEFAULT AND SITE-SPECIFIC IEUBK MODEL PREDICTIONS





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**SECTION 6
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APPENDIX A
GARDEN VEGETABLE AND SOIL DATA

APPENDIX A GARDEN VEGETABLE AND SOIL DATA

Property ID	Garden Vegetables							Garden Soils (mg/kg)		
	Sample ID	Vegetable Type	Dry Wt. Conc (mg/kg dw)		% Solid	Wet Wt. Conc (mg/kg ww)		Sample ID	As	
			As	Pb		As	Pb		As	Pb
1	3-04156-B	Rhubarb	0.05 U	0.61	8.70	4.35E-03	5.31E-02	3-04156-B	11.0 U	122.9
1	3-04159-B	Chard	0.10 J	0.57	6.52	6.52E-03	3.72E-02	3-04159-B	11.0 U	110.2
1	3-04151-B	Peppers	0.05 U	0.11 J	10.60	5.30E-03	1.17E-02	3-04151-B	11.0 U	152.2
1	3-04166-B	Squash	0.06 J	0.20	3.64	2.18E-03	7.28E-03	3-04166-B	15.0	248.8
1	3-04169-B	Squash	0.05 U	0.05 U	13.70	6.85E-03	6.85E-03	3-04169-B	11.0 U	100.8
1	3-04157-B	Eggplant	0.08 J	0.05 U	10.10	8.08E-03	5.05E-03	3-04157-B	11.0 U	127.3
1	3-04158-B	Cabbage	0.05 U	0.05 U	10.10	5.05E-03	5.05E-03	3-04158-B	11.0 U	111.2
1	3-04154-B	Cauliflower	0.05 U	0.05 U	9.98	4.99E-03	4.99E-03	3-04154-B	11.0 U	100.8
1	3-04155-B	Tomatoes	0.05 U	0.05 U	7.71	3.86E-03	3.86E-03	3-04155-B	11.0 U	104.8
1	3-04162-B	Squash	0.05 U	0.07 J	4.26	2.13E-03	2.98E-03	3-04162-B	11.0 U	222.6
2	3-04602-B	Tomatillo	0.05 U	0.05 U	16.50	8.25E-03	8.25E-03	3-04602-B	11.0 U	114.0
3	3-04600-B	Collard Greens	0.34	0.24	12.90	4.39E-02	3.10E-02	3-04600-B	11.0 U	95.9
4	3-04620-B	Lettuce	0.10 J	2.20	10.50	1.05E-02	2.31E-01	3-04620-B	11.5	128.4
4	3-04618-B	Carrots	0.06 J	0.96	13.20	7.92E-03	1.27E-01	3-04618-B	11.0 U	119.2
4	3-04615-B	Beets	0.05 U	0.94	12.70	6.35E-03	1.19E-01	3-04615-B	11.3	129.7
4	3-04617-B	Turnip Greens	0.08 J	0.68	13.60	1.09E-02	9.25E-02	3-04617-B	11.0	130.2
4	3-04619-B	Rutabaga	0.17	0.80	11.40	1.94E-02	9.12E-02	3-04619-B	11.0 U	116.4
4	3-04614-B	Collard Greens	0.32	0.20	17.10	5.47E-02	3.42E-02	3-04614-B	11.0 U	115.5
5	3-04625-B	Collard Greens	0.16	0.50	12.10	1.94E-02	6.05E-02	3-04625-B	11.0 U	52.0 U
5	3-04623-B	Peppers	0.05 U	0.05 U	15.00	7.50E-03	7.50E-03	3-04623-B	12.3	87.3
6	3-04749-B	Onions	6.30	1.78	15.60	9.83E-01	2.78E-01	3-04749-B	73.3	145.5
6	3-04768-B	Carrots	0.50	1.34	13.10	6.55E-02	1.76E-01	3-04768-B	46.5 J	103.9 J
6	3-04758-B	Beets	1.09	1.13	13.90	1.52E-01	1.57E-01	3-04758-B	54.5	98.1
6	3-04755-B	Turnips	3.45	1.21	6.11	2.11E-01	7.39E-02	3-04755-B	40.4	89.7
6	3-04753-B	Cauliflower	0.46	0.50	10.00	4.60E-02	5.00E-02	3-04753-B	92.5	123.6
6	3-04762-B	Collard Greens	0.37	0.12 J	15.50	5.74E-02	1.86E-02	3-04762-B	56.6	140.4
6	3-04756-B	Collard Greens	0.63	0.11 J	16.20	1.02E-01	1.78E-02	3-04756-B	43.0	131.5
6	3-04757-B	Cucumbers	2.92	0.27	5.92	1.73E-01	1.60E-02	3-04757-B	68.6	172.1
6	3-04745-B	Zucchini	1.63	0.11 J	9.54	1.56E-01	1.05E-02	3-04745-B	45.1	280.3
6	3-04743-B	Squash	0.63	0.08 J	9.70	6.11E-02	7.76E-03	3-04743-B	46.2	137.0
6	3-04748-B	Tomatoes	0.08 J	0.05 U	13.30	1.06E-02	6.65E-03	3-04748-B	24.5	138.5
6	3-04769-B	Cabbage	0.31	0.05 U	9.95	3.08E-02	4.98E-03	3-04769-B	48.2	110.1
7	3-05234-B	Cabbage	0.08 J	0.08 J	19.90	1.59E-02	1.59E-02	3-05234-B	11.0 U	65.8
7	3-05225-B	Tomatillo	0.05 U	0.06 J	16.30	8.15E-03	9.78E-03	3-05225-B	11.0 U	56.6

Property ID	Garden Vegetables							Garden Soils (mg/kg)		
	Sample ID	Vegetable Type	Dry Wt. Conc (mg/kg dw)		% Solid	Wet Wt. Conc (mg/kg ww)		Sample ID		
			As	Pb		As	Pb		As	Pb
8	3-05239-B	Beets	0.34	2.32	19.70	6.70E-02	4.57E-01	3-05239-B	11.0 U	236.0
8	3-05240-B	Turnips	0.52	0.98	7.29	3.79E-02	7.14E-02	3-05240-B	19.5	260.5
9	3-05237-B	Tomatoes	0.05 U	0.05 U	7.07	3.54E-03	3.54E-03	3-05237-B	11.0 U	137.2
9	3-05238-B	Tomatoes	0.05 U	0.05 U	6.54	3.27E-03	3.27E-03	3-05238-B	37.1	170.6
10	3-04585-B	Tomatoes	0.05 U	0.62	7.67	3.84E-03	4.76E-02	3-04585-B	18.4	314.1
11	3-04792-B	Garlic	1.24	38.60	16.50	2.05E-01	6.37E+00	3-04792-B	11.0 U	270.6
11	3-05226-B	Chard	0.14 J	4.31	13.20	1.85E-02	5.69E-01	3-05226-B	12.0	147.5
11	3-05222-B	Onions	0.10 J	1.67	12.40	1.24E-02	2.07E-01	3-05222-B	11.0 U	250.1
11	3-05230-B	Collard Greens	0.07 J	0.53	13.80	9.66E-03	7.31E-02	3-05230-B	17.7	184.9
11	3-04791-B	Collard Greens	0.12 J	0.16	13.60	1.63E-02	2.18E-02	3-04791-B	16.4	212.7
11	3-04799-B	Cucumbers	0.67	0.18	6.02	4.03E-02	1.08E-02	3-04799-B	16.7	259.1
12	3-04773-B	Carrots	0.27	1.15	11.00	2.97E-02	1.27E-01	3-04773-B	26.0	140.1
12	3-04765-B	Collard Greens	0.38	0.58	12.00	4.56E-02	6.96E-02	3-04765-B	17.5	224.9
12	3-04776-B	Collard Greens	0.56	0.30	13.00	7.28E-02	3.90E-02	3-04776-B	22.8	157.4
12	3-04775-B	Collard Greens	0.27	0.29	11.00	2.97E-02	3.19E-02	3-04775-B	25.1	152.4
13	3-04789-B	Onions	0.17	1.87	14.80	2.52E-02	2.77E-01	3-04789-B	16.9	217.7
13	3-04798-B	Celery	0.19	2.05	8.25	1.57E-02	1.69E-01	3-04798-B	11.0 U	338.8
13	3-04794-B	Turnips	0.33	1.57	10.20	3.37E-02	1.60E-01	3-04794-B	11.0 U	210.3
13	3-04779-B	Collard Greens	0.11 J	0.16	13.70	1.51E-02	2.19E-02	3-04779-B	25.4	344.0
13	3-04786-B	Squash	0.05 J	0.29	6.17	3.09E-03	1.79E-02	3-04786-B	11.0 U	240.8
13	3-04782-B	Peas	0.05 U	0.06 J	22.50	1.13E-02	1.35E-02	3-04782-B	11.0 U	294.0
13	3-04771-B	Cabbage	0.13 J	0.05 U	11.60	1.51E-02	5.80E-03	3-04771-B	11.5	186.0
13	3-04784-B	Tomatoes	0.05 U	0.05 U	9.96	4.98E-03	4.98E-03	3-04784-B	11.0 U	253.0
13	3-04781-B	Cabbage	0.05 U	0.05 U	9.24	4.62E-03	4.62E-03	3-04781-B	11.0 U	195.5
14	3-04148-B	Onions	0.14 J	0.68	13.80	1.93E-02	9.38E-02	3-04148-B	11.6	162.2
14	3-04144-B	Peppers	0.05 U	0.20	9.68	4.84E-03	1.94E-02	3-04144-B	15.3	171.6
14	3-04150-B	Broccoli	0.08 J	0.06 J	12.20	9.76E-03	7.32E-03	3-04150-B	21.3	183.5
15	3-05249-B	Cucumbers	0.68	0.66	4.55	3.09E-02	3.00E-02	3-05249-B	11.0 U	369.3
15	3-05247-B	Tomatoes	0.05 U	0.33	5.84	2.92E-03	1.93E-02	3-05247-B	11.0 U	570.0
15	3-05248-B	Tomatillo	0.10 J	0.16	7.37	7.37E-03	1.18E-02	3-05248-B	11.0 U	335.1
15	3-05244-B	Tomatoes	0.05 U	0.10 J	6.63	3.32E-03	6.63E-03	3-05244-B	11.0 U	381.1
16	3-04608-B	Peppers	0.15	0.15 J	13.10	1.97E-02	1.97E-02	3-04608-B	15.2	79.5
17	3-04588-B	Tomatoes	0.05 U	0.18	5.90	2.95E-03	1.06E-02	3-04588-B	11.0 U	52.0 U
17	3-04589-B	Peppers	0.05 U	0.05 U	13.60	6.80E-03	6.80E-03	3-04589-B	11.0 U	61.2
18	3-04744-B	Tomatoes	0.05 U	0.11 J	5.77	2.89E-03	6.35E-03	3-04744-B	13.7	572.9
19	3-04597-B	Beans	0.05 U	0.13 J	19.00	9.50E-03	2.47E-02	3-04597-B	11.0 U	408.3
19	3-04595-B	Tomatillo	0.05 U	0.20	6.47	3.24E-03	1.29E-02	3-04595-B	16.0	236.1
19	3-04592-B	Tomatoes	0.05 U	0.05 U	6.05	3.03E-03	3.03E-03	3-04592-B	11.0 U	260.8

APPENDIX B

**SCREENING LEVEL EVALUATION OF
RELATIVE RISK FROM ARSENIC VIA
INHALATION OF DUST OR DERMAL CONTACT WITH SOIL
COMPARED TO SOIL INGESTION**

APPENDIX B

SCREENING LEVEL EVALUATION OF RELATIVE RISK FROM ARSENIC VIA INHALATION OF DUST OR DERMAL CONTACT WITH SOIL COMPARED TO SOIL INGESTION

1.0 INHALATION OF PARTICULATES IN AIR

The basic equations recommended by USEPA (1989) for evaluation of risk from inhalation exposure of soil particles in air and for incidental ingestion of soil are as follows:

Inhalation Exposure

$$\text{Risk}_{\text{air}} = C_a \cdot BR_a \cdot EF \cdot ED / (BW \cdot AT) \cdot SF_{\text{inh}}$$

Ingestion Exposure

$$\text{Risk}_{\text{soil}} = C_{\text{soil}} \cdot IR_{\text{soil}} \cdot EF \cdot ED / (BW \cdot AT) \cdot SF_{\text{oral}}$$

where:

- C = Concentration of contaminant in air (C_a , mg/m³) or soil (C_{soil} , mg/kg)
- BR = Breathing rate (m³/day)
- IR_{soil} = Ingestion rate for soil (kg/day)
- EF = Exposure frequency (days/yr)
- ED = Exposure duration (years)
- BW = Body weight (kg)
- AT = Averaging time (days)
- SF = Cancer slope factor for inhalation or oral exposure

Assuming that the values of BW, EF, ED, and AT are all the same for inhalation and oral exposure, the ratio of the risk from inhalation of particulates in air to that from ingestion of soil is then:

$$\text{Relative risk (inhalation/oral)} = (C_{\text{air}}/C_{\text{soil}})(BR/IR)(SF_{\text{inhal}}/SF_{\text{oral}})$$

Screening level defaults inputs for this equation are as follows:

- The ratio $C_{\text{air}}/C_{\text{soil}}$ is given by the EPA recommended default Particulate Emission Factor (PEF) of 7.6E-10 kg/m³ (EPA 1996)
- The ratio of BR/IR for a resident is 20 m³/day / 1E-04 kg/day = 2E+05 m³/kg (USEPA 1989, 1991b)

- For arsenic, the ratio of the inhalation slope factor to the oral slope factor is 15/1.5 = 10 (IRIS 2000).

Based on these values, the ratio of the risk from inhalation exposure to arsenic in airborne soil particles compared to that from ingestion exposure is:

$$\text{Relative risk} = 7.6\text{E-}10 \cdot 2\text{E+}05 \cdot 10 = 0.00152 \text{ (0.15\%)}$$

As seen, the risk from inhaled arsenic is very small (< 0.2%) compared to that from ingested soil, so this pathway is considered to be sufficiently minor that quantitative evaluation is not required at this site.

2.0 DERMAL EXPOSURE VIA SOIL

The basic equations recommended for estimation of risk from dermal contact with soil and ingestion of soil are as follows (EPA 1989, 1992):

Dermal Exposure

$$\text{Risk}_{\text{dermal}} = C_s \cdot SA \cdot AF \cdot ABS \cdot EF \cdot ED / (BW \cdot AT) \cdot (SF_{\text{oral}} / AF_o)$$

Oral Exposure

$$\text{Risk}_{\text{soil}} = C_s \cdot IR_{\text{soil}} \cdot EF \cdot ED / (BW \cdot AT) \cdot SF_{\text{oral}}$$

where:

C_s	=	concentration of chemical in soil (mg/kg)
SA	=	surface area in contact with soil (cm ²)
AF	=	soil adherence factor (kg/cm ²)
ABS	=	dermal absorption fraction (unitless)
AF_o	=	oral absorption fraction
IR_{soil}	=	ingestion rate for soil (kg/day)
BW	=	body weight (kg)
EF	=	exposure frequency (days/yr)
ED	=	exposure duration (years)
AT	=	averaging time (days)
SF_{oral}	=	cancer slope factor for oral exposure

Thus, assuming the values of BW, ED, and AT are the same for dermal and oral exposure, the ratio of the risk for dermal contact compared to that for soil ingestion is given by:

$$\text{Relative risk (dermal/oral)} = (SA \cdot AF \cdot EF_{\text{dermal}} \cdot ABS) / (IR \cdot EF_{\text{oral}} \cdot AF_o)$$

Screening level inputs for this equation are as follows:

- $SA = 10\%$ of whole body = 2,000 cm² (USEPA 1991b).

- $AF = 1E-06 \text{ kg/cm}^2$ (USEPA 1992)
- $EF_{\text{dermal}} = 50 \text{ days/yr}$ (assumed)
- ABS is not known for arsenic, but is likely to be no higher than 0.01 (USEPA 1992)
- $IR = 1E-04 \text{ kg/day}$ (USEPA 1989, 1991b)
- $EF_{\text{oral}} = 350 \text{ days/yr}$ (USEPA 1989, 1991b)
- $AFo = 1.0$ for arsenic (assumed)

Based on these inputs, the estimated ratio of dermal risk to ingestion risk for arsenic in soil is:

$$\text{Relative Risk} = (2E+03 \cdot 1E-06 \cdot 50 \cdot 0.01) / (1E-04 \cdot 350 \cdot 1.0) = 0.029 \text{ (2.9\%)}$$

Thus, the relative risk from dermal contact with arsenic in soil compared to ingestion exposure is likely to be no more than about 3 %, and could be less if the frequency or extent of dermal contact is lower than assumed, or if the dermal absorption fraction for arsenic is lower than 0.01. On this basis, it is concluded that dermal absorption is a minor contributor of risk compared to oral exposure, and that this pathway may be excluded from quantitative evaluation.

3.0 REFERENCES

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APPENDIX C
RISK-BASED CONCENTRATION VALUES
FOR WORKERS

APPENDIX C

RISK-BASED CONCENTRATION VALUES FOR WORKERS

1.0 OVERVIEW

A Risk-Based Concentrations (RBC) is a concentration of a chemical in a medium that is not of health concern to a specified population under a specified set of exposure assumptions. RBC values are derived by reversing the risk assessment process, solving for the concentration of a chemical that corresponds to a specified target risk value. This Appendix calculates the RBC values for exposure of workers to arsenic and lead in soil. These values may then be used to assess whether there is a need for quantitative evaluation of risk to this population.

2.0 RBC FOR ARSENIC

The basic equation used to calculate the RBC for exposure of workers to arsenic in soil is:

$$RBC = \frac{\text{Target Risk}}{\left(\frac{IR}{BW}\right)\left(\frac{EF \cdot ED}{AT}\right)(oSF \cdot RBA)}$$

Input values applicable to worker exposure to soil are listed below, along with the resulting RBC value.

Parameter	Default Value	Source
Target Risk	1E-04	USEPA 1991b
IR (kg/day)	1E-04	USEPA 1991a
BW (kg)	70	USEPA 1991a
EF (days/yr)	250	USEPA 1991a
ED (years)	25	USEPA 1991a
AT (years)	70	USEPA 1991a
RBA	0.5	USEPA 2000
oSF (mg/kg-d) ⁻¹	1.5	IRIS 2000
RBC (mg/kg)	382	Calculated

3.0 RBC FOR LEAD

The EPA has not established a default soil action level for lead for protection of workers. However, the EPA has developed an interim method for calculating the risk to workers from lead in soil (USEPA 1996). The basic equation is:

$$GM\ PbB = PbB0 + PbS \cdot BKSF \cdot IR_s \cdot AF_s \cdot EF_s / AT$$

where:

GM PbB =	Geometric mean blood lead (ug/dL) in a population of workers
PbB0 =	Baseline geometric mean blood lead value (ug/dL) in the workers in the absence of occupational exposure
BKSF =	Biokinetic slope factor (ug/dL increase in blood lead per ug/day of lead absorbed)
PbS =	Concentration of lead in soil (ug/g)
IR _s =	Intake rate of soil (g/day)
AF _s =	Absorption fraction for lead from soil. This value is given by: $AF_s = AF_{food} \cdot RBA_{soil}$
EF _s =	Exposure frequency to soil (days/yr)
AT =	Averaging time (days)

Given the GM PbB, and assuming the distribution of PbB values is lognormal with a geometric standard deviation of GSD, the 95th percentile of the distribution is given by:

$$95th = GM \cdot GSD^{1.645}$$

The subpopulation of primary concern for protection of workers from excessive lead exposure is pregnant females. The goal is to ensure that there is no more than a 5% chance that the blood lead level of the fetus will exceed 10 ug/dL. The ratio between the blood lead concentration in the mother and the fetus is given by:

$$R(\text{fetal/maternal}) = PbB(\text{fetus}) / PbB(\text{mother})$$

Default input values recommended by EPA for each of these parameters are summarized in Table C-1. Using these inputs, the concentration of lead in soil which yields a 95th percentile value of 10 ug/dL in the blood of the fetus may be calculated. This value is 1,545 ppm.

**TABLE C-1 DEFAULT INPUTS
ADULT WORKER LEAD EXPOSURE MODEL**

INPUTS

PbB0	2.0 ug/dL
BKSF	0.4 ug/dL per ug/day
IRsoil	0.05 g/day
EFsoil	219 days/yr
AT	365 days/yr
AFfood	0.2
RBAsoil	0.6
R(fetal/maternal)	0.9
GSD	1.8

CALCULATED VALUES

Target 95th (maternal)	11.1 ug/dL
Target GM (maternal)	4.23 ug/dL
AFsoil	0.12

RESULT

RBC	1545 ug/g
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CHECK

GM	4.23 ug/dL
95th	11.11 ug/dL